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CLAUD WORTH

FIFTH EDITION

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SQUINT:

ITS CAUSES, PATHOLOGY, AND TREATMENT

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SQUINT

ITS CAUSES, PATHOLOGY, AND TREATMENT

BY

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PREFACE TO THE FIFTH EDITION

THE First Edition of this book was not published until 1903, when I thought that I could be certain of what I said; subsequent editions, therefore, have not greatly differed from the first, though some improvements have been made.

Of the cases of squint in which efficient treatment is carried out from the first appearance of the deviation, only a small proportion will ever need operation.

Operation is required in the majority of the neglected or inefficiently treated cases. A great advance here is the abolition of the "combined operation." Formerly, though recognising the danger and uncertainty of simple tenotomy, I thought that a convergence of high degree and long standing might safely be remedied by tenotomy of an internal rectus combined with advancement of the externus. The following statement shows how experience gradually led me to modify my practice in this respect:—

I have endeavoured, as far as possible, to keep in touch with my squint operation cases in private practice up to the present date, as well as with many of my early hospital cases. The patients are re-

examined (those abroad are written to) at intervals, which, in the older cases, may be as long as three or four years. I have arranged the cases in three groups: (1) Those before the end of December, 1901. A period of nearly twenty years has elapsed since the most recent of these operations. Besides simple advancements (by the method described in Chapter XII.), this group includes advancements combined with tenotomy of the opponent muscle, shortening of muscles, and simple tenotomies. (2) Operations between December, 1901, and December, 1905. This group contains mostly simple advancements, some tenotomies combined with advancement, and scarcely any simple tenotomies. (3) Cases since December, 1905. This group consists exclusively of simple advancements. This advancement operation admits of accurate adjustment, and the results have proved to be permanent.

LONDON,
February, 1921.

PREFACE TO THE FIRST EDITION

By examining a very large number of cases of squint, and watching the results of treatment during a number of years, and by investigating the visual functions of normal-sighted people, I have endeavoured to learn the causes and pathology of squint. The methods of treatment which I employ are the outcome of these observations.

In cases of constant unilateral convergent squint, the usual routine treatment, by glasses and operation, gives extremely unsatisfactory results. In about one-third of these cases the wearing of glasses causes the eyes, after a time, to become "straight." In the other two-thirds the *deformity* may be more or less removed by operation. But, more often than not, the deviating eye becomes very blind, and the acquisition of any sort of binocular vision is quite the exception.

On the other hand, cases of unilateral squint in which treatment is commenced early and carried out by the methods described in these pages are nearly always perfectly cured, having good vision in each eye, and good binocular vision.

Since 1893 I have kept detailed notes of every case of squint which has come under my observation.

I have notes of 2,337 squints and heterophorias. Of these cases, 1,729 suffered from convergent squint. The cases presented themselves in the out-patient departments of the West Ham and East London Hospital and the Royal London Ophthalmic Hospital (Moorfields) and in my private practice.

I am greatly indebted to Mr. Silcock and Mr. Holmes Spicer for their kindness in allowing me, during a period of nearly four years, to investigate and treat the cases of squint attending their out-patient clinics at Moorfields.

I wish to thank Mr. R. E. Hanson for valuable assistance in working out the statistics of my cases.

HARLEY STREET,
LONDON, W.,
May, 1903.

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SQUINT:

ITS CAUSES, PATHOLOGY AND TREATMENT

CHAPTER I

INTRODUCTION

THIS chapter contains nothing new. It deals very briefly with certain elementary facts.

EMMETROPIA is the refractive condition of the normal adult human eye. Rays of light proceeding from a single point on a distant object may, for practical purposes, be regarded as parallel. When these parallel rays enter an emmetropic eye, they undergo refraction as they pass through the refracting media (cornea, aqueous humour, lens, vitreous humour), and are brought to a focus on the retina. Rays from every other point in the distant object are similarly focussed, so that a complete (inverted) image of the object is formed on the retina. The refraction which takes place under these conditions is called the *static refraction* of the eye.

ACCOMMODATION.—Rays of light which enter the eye from a near object, *e.g.*, a printed page, are sensibly divergent. Now it is obvious that the *static* refraction of the normal eye, which exactly suffices to bring parallel rays to a focus on the retina, will not accurately focus these divergent rays. To meet this deficiency, there is a muscle within the eyeball, the

ciliary muscle, which, by its contraction, causes the lens to become more convex, more nearly spherical, and so increases its refractive power. This act of increasing the refractive power of the eye is called *accommodation*, and the additional refraction thus produced is called the *dynamic refraction* of the eye.

PRESBYOPIA.—In childhood the lens is very soft and elastic, and is easily made to change its shape under the action of the ciliary muscle, so that children have a very wide range of accommodation. As age advances, the lens gradually becomes more and more firm and incompressible, so that, though distant objects are still perfectly focussed by means of the static refraction of the eye, the increase of refraction, produced by the action of the ciliary muscle on the lens, becomes, after a time, insufficient for the focussing of near objects. For this reason, a normal-sighted person of fifty must either hold his book at a greater distance, in order that the rays of light proceeding therefrom shall be more nearly parallel, or he must supplement his weakened accommodation with a pair of convex glasses.

ATROPINE, when instilled into the conjunctival sac, has the property of temporarily paralysing the ciliary muscle, and so suspending entirely the power of accommodation. An atropised normal eye sees distant objects distinctly, by virtue of its static refraction, but is quite unable to focus the divergent rays proceeding from a near object. In other words, atropine produces an artificial presbyopia.

FIXATION.—In the centre of the retina is the macula lutea, which, in the human eye, is far more sensitive to ordinary visual impressions than any other part. It is desirable, therefore, that the eye be brought into such a position that the image of any object which especially engages our attention shall be formed upon the macula lutea. The eye is then said to "fix" the object. An imaginary line, passing from the centre

of the macula, through the optical centre of the eye, to the object looked at, is called the *visual axis*.

CONVERGENCE.—When the two eyes look at a distant object, the visual axes may, for practical purposes, be considered to be parallel. When, however, a near object is looked at, the two eyes must rotate inwards, in order that both visual axes may be directed to the same object. This active inward rotation of the eyes is called *dynamic convergence*. In the case of a normal pair of eyes there is no such thing as *static convergence*, because the primary position of the visual axes is one of parallelism. In a case of convergent squint, however, there is a static convergence corresponding to the angle of the deviation.

ACCOMMODATION AND CONVERGENCE.—When a person with a normal pair of emmetropic eyes looks at a near object, the eyes converge in order that both visual axes may be directed to the object. At the same time each eye "accommodates," in order that the rays of light from the object may be accurately focussed on its retina. These two functions, accommodation and convergence, are, in ordinary life, always used together, so that they have become "associated" by hereditary and individual habit. It is difficult, therefore, for a normal pair of eyes to accommodate without converging or to converge without accommodating.

CONJUGATE MOVEMENTS.—In looking to the right, or left, or up, or down, the two eyes move together through exactly the same angle.

MOVEMENTS OF EACH EYE SEPARATELY.—The extreme range of upward and downward rotations of a single eye varies slightly in different people, the average being about 46° up and 56° down.[1] Outward

[1] These figures are the average of measurements which I made on 64 normal-sighted persons with Stephens' tropometer.

rotation (abversion) may be considered full when the edge of the cornea can be made to touch the outer canthus. The power of inward rotation (adversion) varies considerably in different people. Most people can advert each eye separately through an arc of 50°. The power of independent adversion tends to become less as age advances.

HYPERMETROPIA.—For purposes of discussion, the refracting media of the eye may be diagrammatically represented as a simple convex lens. In the emmetropic eye, as already explained, the strength of this lens is such that parallel rays of light are brought to a focus exactly at the retina. A hypermetropic eye is shorter, from before backwards, than the emmetropic eye. This abnormal shortness causes the retina to be situated too near this diagrammatic lens. Now, in order that parallel rays may be brought to a focus on this abnormally situated retina, the focal length of the diagrammatic lens must be shortened, or, in other words, the strength of the lens must be increased. This increase in strength may be brought about either by the patient's using his accommodation in distant vision (and still more, of course, in near vision) or by his wearing a convex spectacle lens in front of the eye. A hypermetrope may be able easily to accommodate sufficiently to correct his refractive error in distant vision, but may have difficulty in sustaining the additional effort of accommodation involved in looking at a near object, *e.g.*, in reading.

MYOPIA is the converse of hypermetropia. A myopic eye is abnormally long from before backwards, so that the retina is at a greater distance from the centre of our diagrammatic lens than is the case in the emmetropic eye. In order, therefore, that parallel rays from a distant object shall be focussed on the abnormally situated retina, the focal length of the diagrammatic lens must be increased, *i.e.*, the strength of the lens must be diminished. The only way in which this

can be accomplished is by putting a concave spectacle lens in front of the eye.

If an indistinctly seen distant object be gradually brought nearer a myopic eye, the rays which enter the eye therefrom become more and more divergent, till a point is reached at which the static refraction of the myopic eye is just sufficient to focus these divergent rays. If the object be brought still nearer, the eye accommodates and so still sees distinctly.

ASTIGMATISM.—In the human eye, the greater part of the refraction takes place at the surface of the cornea, where the rays of light pass from the air into the much denser medium, the corneal substance. The normal human cornea is approximately a segment of a sphere. Sometimes, however, the cornea is curved more in one meridian than in another, so that it is slightly oval—like a slice from the side of a cocoa-nut. This condition is called astigmatism. In the meridian of greater curvature the rays will be refracted more, and brought to a focus sooner, than in the meridian of less curvature. Consequently the picture formed on the retina will be blurred and indistinct. In order to equalise the refraction in the different meridians a cylindrical lens must be used, *i.e.*, a lens which is curved in one direction only.

ANISOMETROPIA is an inequality in the static refraction of the two eyes.

ANGLE GAMMA.[1]—The visual axis does not exactly correspond with the geometrical antero-posterior axis

[1] The angle gamma which I have described is the angle which is measured clinically and is of practical importance. But, since the visual axis need not necessarily pass through the centre of motion of the eyeball, mathematicians have taken as one of the boundaries of the angle gamma a line passing through the centre of motion of the eyeball to the object looked at. This line is of no clinical significance, as its direction cannot be determined except on paper.

of the eyeball. The angle between the visual axis and the antero-posterior axis of the eyeball is called the *angle gamma*. Usually the visual axis passes through the pupil to the nasal side of its centre. Rarely it passes through the pupil to the temporal side of its centre, in which case the angle gamma is said to be negative.

In hypermetropic eyes the angle gamma is usually high, so that, when the visual axes are parallel, the antero-posterior axes of the eyeballs are perceptibly divergent. In this way a deceptive appearance of divergent squint may be produced or a slight convergent squint may be masked.

In myopic eyes the angle gamma usually is low or even negative. In the latter case, while the visual axes are parallel, the antero-posterior axes of the eyeballs are convergent, so that convergent squint may be simulated or a slight divergent squint masked.

CHAPTER II

BINOCULAR VISION

WHEN the eyes are in the primary position (*i.e.*, looking straight ahead into the distance) the fields of vision of the two eyes overlap everywhere, except in a sector of about 35° towards the temporal periphery of each field. In other words, everything which a normal-sighted person sees, he sees with both eyes simultaneously, except objects which lie on his extreme right or his extreme left. These are seen only with one eye.

When a distant object engages our attention, the two eyes are brought into such a position that a picture of the object is formed simultaneously on the central part of each retina. Similarly, all other distant objects, within the limits of overlapping of the visual fields, are focussed on functionally corresponding parts of each retina. The impressions thus received from the two eyes are blended in the brain, so that we are conscious only of one single picture. This psychical blending of the two sets of visual impressions is called binocular vision.

The binocular vision of near objects is a more

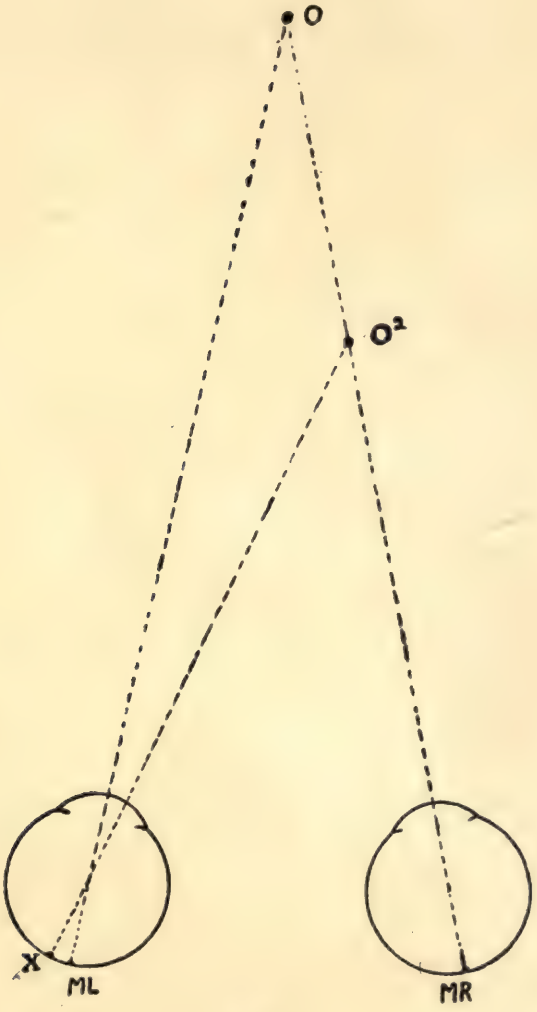

FIG. 1.

complex act, inasmuch as the brain has to blend images which, for the most part, do not fall upon geometrically corresponding points of the two retinæ.

The diagram represents a pair of eyes looking at an object O in the middle distance. An image of O is formed on the macula ML and MR of each retina. Now, it is obvious that only objects lying in the line MR—O (or in this line produced beyond O) can be focussed on the macula of the right eye. Similarly, only objects lying in the line ML—O can be focussed on the macula of the left eye. O^2 is an object lying in the line MR—O. An image of O^2 will be formed on the macula of the right eye. But the image of O^2 formed in the left eye will not be on the corresponding point, but at a point X, considerably to the outer side of the macula.

Take a practical illustration—look steadily at a distant object, and hold a finger about 18 inches in front of the eyes. The finger will be seen double, the left image corresponding to the right eye, and the right image to the left eye (crossed diplopia). Now look at the finger, and the distant object will appear double, the diplopia this time being homonymous.

This "physiological diplopia" must be constantly present, in looking about a room for example, yet we are not ordinarily conscious of seeing double. This customary freedom from

diplopia is brought about, not by mental suppression of one of the images, but by the marvellous elasticity of the fusion faculty. Both sets of impressions reach the brain, and, by their combination, assist in our appreciation of the third dimension.

A simple experiment will demonstrate the elasticity of fusion. Place in the amblyoscope (chapter viii.) the slides shown in fig. 14, p. 126. The two slits are fused into one, and the control marks are seen, one on each side. Now gradually diverge the tubes. When the extreme limit of divergence of the visual axes has been nearly reached, fusion of the slits is still maintained, but the control marks recede on each side farther and farther from them. When the limit is passed and fusion can no longer be maintained, the slits suddenly spring apart. As each slit is on the same slide with its control mark, the distance cannot really vary; but, within certain limits, the mind still fuses the images of the slits, even when they no longer fall upon anatomically corresponding points of each retina. This experiment would serve as a proof, were any needed, that fusion is a purely psychical process, and not merely the result of stimulation of corresponding sets of nerve endings in each retina.

But this must not be taken to indicate that accurate adjustment of the relative positions of the two eyes is unnecessary. The *law* governing the

fusion of images which are not precisely similar may be stated as follows. *When the images formed in the two eyes differ in shape, size, or position, if the disparity be not too great, the oculo-motor apparatus first places the eyes in the most favourable relative positions; the fusion sense, by virtue of its elasticity, then fills up any gap which may remain.*

The following additional experiments were suggested to me by Dr. Verhoeff, of Boston, U.S.A.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

Place fig. 2 and fig. 3 in a stereoscope. The horizontal lines of course are blended. But the oblique lines also are blended; the resulting line appearing perpendicular to the horizontal line. This blending

of the oblique lines cannot be accomplished by any rotation of the eyeballs round a fore-and-aft axis, as, in this case, the horizontal lines would not be blended.

Again, if figs. 4 and 5 be blended in a stereoscope the combined image resembles fig. 6. The oblique lines outside the circles are blended into a perpendicular line, but the part within the circle, which is seen with one eye only, retains its oblique direction.

In a case in which each eye separately has the power of seeing, but in which binocular vision is absent, one of two conditions must be present: either (*a*) the mind is separately conscious of the two sets of impressions received from the two eyes —diplopia, *e.g.*, in paralysis of an external ocular muscle; or (*b*) the mind takes note only of the impressions received from one eye and ignores those received from the other—suppression, *e.g.*, in an ordinary case of convergent squint.

Grades of Binocular Vision.

Most people who have binocular vision have the faculty to its full extent. Anyone, however, who has undertaken the orthoptic training of any considerable number of squinters will find that those who see binocularly naturally arrange themselves into three separate and distinct classes, according to the degree in which they possess the faculty.

These may be called:—

First Grade.—Simultaneous macular perception.

Second Grade.—True fusion with some amplitude.

Third Grade.—Sense of perspective.

First Grade. Simultaneous macular perception.—A patient having only this grade of binocular vision sees devices in a stereoscope as two separate pictures, which overlap and form one only when they are put in certain relative positions corresponding to the directions independently assumed by the visual axes. The "desire" for binocular vision is absent, so that no effort will be made to maintain fusion.

Second Grade. True fusion with some amplitude.—A person having the second grade of binocular vision not only fuses the retinal images in the two eyes, but can make some effort to maintain fusion. When such a person is fusing the pictures in a stereoscope, if the pictures be separated or brought together the eyes will, to a certain extent, follow them in the interest of binocular vision.

Third Grade. Sense of perspective.—The two eyes see from different points of view. In looking at any solid object, such as a pillar, for instance, the right eye will see more of the right side of the object and the left eye more of the left side. In the slightly dissimilar pictures thus focussed on the retinæ the points of difference are not suppressed, as in the case of a person having only the second grade, neither is the observer conscious of

diplopia. The psychical blending of the two slightly dissimilar sets of visual impressions enables him to appreciate the solidity of surrounding objects and assists in his judgment of their relative distances.

There is a wide gap between grades 1 and 2. A patient, however, who has grade 2 usually acquires the third grade also.

Quite as important as the grade of binocular vision is its intensity. A person whose fusion sense is feebly developed may possibly, under favourable conditions, have the highest grade of binocular vision. But the intensity of his tendency to fusion will be slight, so that, under unfavourable conditions, he easily abandons the effort and uses one eye only. One, however, whose fusion sense is well developed will have such an intense tendency to binocular vision that nothing will make him abandon it while both eyes are open. (Except, of course, a muscular paralysis, in which case he will suffer from persistent and intolerable diplopia.)

Tests for Binocular Vision.

Four-dot test.—A convenient clinical test, which I have used constantly for some years, is an adaptation of Snellen's coloured glasses. For want of a better name, it may be called the "Four-dot test." A pure red glass allows only the red rays of light to pass through it. A pure green glass

transmits only the green rays. Therefore light which has passed through the red glass cannot be seen at all through the green glass, and *vice versa*.

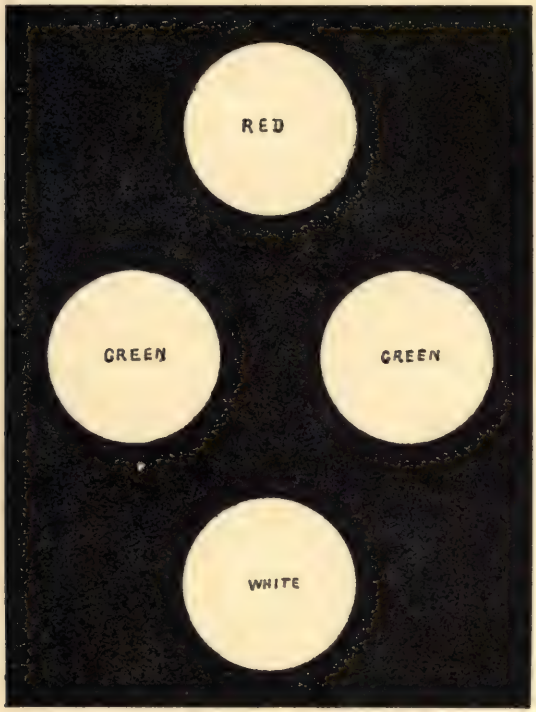

FIG. 7.

A piece of plain ground glass, 12 inches by 9 inches, is covered on the back with opaque black paper. The black paper has four round holes cut in it, each 3 inches in diameter, as shown in the diagram. The lower hole is left clear. Behind the upper hole is cemented a piece of red glass.

Behind each of the other two is cemented a piece of green glass. The arrangement is mounted in the front of a box which contains an electric or other bright light.

The patient, standing five or six yards away, wears a trial frame with a red glass before the right eye and a green glass before the left. If now he sees two dots (white and red) he is using the right eye only. If he sees three dots (white and two green) he is using the left eye only. If he sees four dots (white, red, and two green) he uses both eyes, and has at least grade 1 binocular vision. If he sees five dots (red, two green, and the white seen double) he has diplopia. If the accuracy of the patient's answers be doubted, it may be tested by changing the glasses in the spectacle frame from one eye to the other.

The amblyoscope (see chapter viii.).—Adjust the instrument for parallelism of the visual axes. Place in the slots the slides shown in fig. 14. If the patient sees the two slits as one, and, at the same time, sees both the dot and the cross, he has grade 1 binocular vision. Now diverge or converge the tubes of the instrument. If this can be done, even to a very slight extent, while the patient still fuses the slits and sees both control marks, he has grade 2 binocular vision.

For children, more interesting objects, such as figs. 17 and 18, may be used in the same way. The extent to which the tubes may be separated

or brought together without the eyes becoming dissociated may, for practical purposes, be taken as a measure of the degree of development of the fusion faculty.

Now put in the instrument the slides shown in fig. 19. A patient with grade 2 binocular vision will only fuse the outer circles; but he will either suppress the image of one of the inner circles, or will see both inner circles "all mixed up." A person, however, with grade 3 binocular vision sees the inner circles blended and appearing much farther from the eye, giving the impression that one is looking into a tub or bucket. If the slides be now changed from one tube to the other, the inner circle will seem nearer the eyes, as if one were looking at a tub bottom upwards. This appearance is so vivid that even young children can tell at once whether they are looking at the outside or the inside of the tub. By changing the slides from one tube to the other two or three times, guessing on the part of the patient is rendered impossible. I know no better test for the "sense of perspective."

Hering's drop test for the sense of perspective.—I do not now use this test clinically. The experiment is, however, very instructive.

The test apparatus (fig. 8) consists of a shallow box (of about the size and shape of a box for twenty-five large cigars), open at both ends. From one end two arms project. The extremities of these two arms are joined by a fine thread, on the middle of which is a round bead.

The patient holds the open end of the box close to his eyes, and looks through the box at the bead. The surgeon now drops small objects of various sizes, such as cowrie shells, sometimes on one side of the thread, sometimes on the other. The patient is asked to say whether each shell, as it drops, falls on the near side or the far side of the thread.

If he has the third grade of binocular vision, "the sense of perspective," he will almost always answer

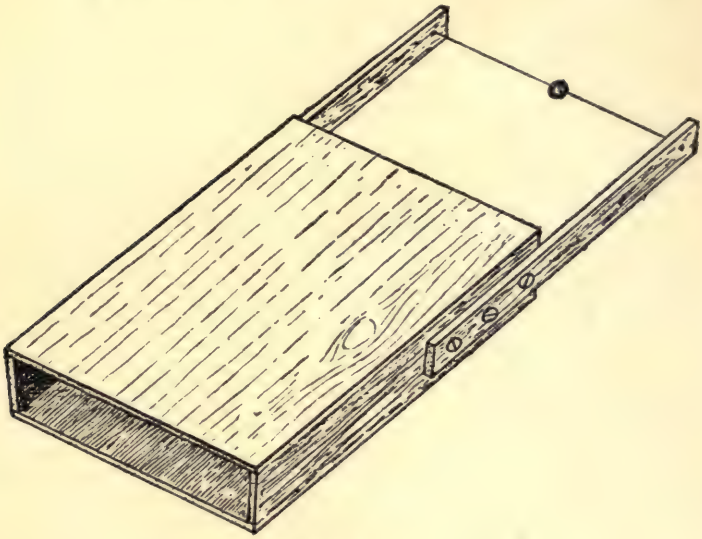

FIG. 8.

correctly. If he has not, his answers will be mere guesses, and he will be nearly as often wrong as right.

The principle of the test is as follows: The box cuts off all view of surrounding objects, including the hands of the surgeon. The size of the falling objects varies, so that their apparent size gives no information as to their distance. The view of the falling object is too brief to admit of any movement of accommodation or convergence, or any lateral movement of the

patient's head. The patient is thus deprived of all auxiliary means of judging distances, and has to depend upon his "sense of perspective" alone.

This test is not infallible. I once made the discovery that a boy, aged 11, with a manifest squint of 13°, could tell on which side the shell fell, almost every time, when both eyes were open. When, however, I covered the deviating eye his answers were as often wrong as right. He had, in the deviating eye, $\frac{6}{18}$ vision. There was no "false macula." He had no diplopia spontaneously, but it was easily elicited with a candle and coloured glasses. I have since met with other similar cases. The explanation is probably this: In trying the test with a normal pair of eyes, the view of the falling shell is so brief that there is no time for the eyes "to fix" the shell, so the images of the object do not fall upon corresponding parts of the two retinæ. It is not, therefore, really a test for the sense of perspective in direct vision, but a test for the subconscious perception of "physiological diplopia" in more or less eccentric parts of the retinæ.

In none of these cases was the suppression of the vision of the deviating eye very profound, so that a moving object would probably be perceived. As strictly corresponding points are not required in this test, it seems not unlikely that the mind may derive some information from the false image, by making allowance for the faulty position of the deviating eye. This is not difficult to believe when we remember that, in a case of squint in which diplopia has been artificially elicited, the angle of the diplopia is often very much smaller than that of the squint; showing that the mind ordinarily makes some allowance for the position of the squinting eye.

The Normal Development of the Fusion Sense.

In two large crêches I made experiments, extending over nearly a year, with the object of gaining some knowledge of this subject. It would be tedious to describe in detail the methods employed. I therefore give a general summary of results.

From the earliest infancy the pupillary light reflex and the fixation reflex are present, showing that some degree of vision of each eye, and the preponderance of the macular region, are innate. If, in a darkened room, the light of a candle be suddenly thrown from an ophthalmoscope mirror into an eye of an infant only a few hours old, the eye will immediately turn towards the mirror. This fixation is purely reflex and is only momentary. During the first few days of life the infant cannot fix a steady light, but, by suddenly flashing the light into the eye, this reflex fixation may be repeatedly obtained.

At the end of two or three weeks most infants will fix the mirror steadily for several seconds at a time with one or other eye, but will not converge both visual axes accurately in looking at a near object.

At the age of five or six weeks, as a rule, the positions of the reflections of the mirror on the child's corneæ are symmetrical, showing that the

child is fixing the mirror binocularly. But, now and then, one eye turns a little inwards, or more rarely outwards, while the other fixes the mirror.

During the first few months of life the movements of the eyes are uncertain, not completely controlled by the higher centres of the brain. The eyes move more or less together, but the slightest gastric or other disturbance often causes one or other eye to deviate. But it will be noticed that this want of co-ordination is confined to movements in the horizontal plane. The conjugation of the two eyes for vertical movements is well developed from the earliest infancy ; one does not see one eye turn up or down without the other.

We can scarcely suppose that any considerable degree of binocular vision can be present at a time when the co-ordination of the eyes in horizontal movements is in this rudimentary condition.

A little later, at the age of five or six months, if the child's attention be engaged by some new and bright object of absorbing interest, such as a cut glass decanter-stopper revolved before a candle flame, it will often be possible for the person nursing the child to slip a large prism before one of his eyes without his appearing to notice it. A prism displaces the image of an object towards its apex. Therefore, if binocular vision is to be maintained, the eye must also rotate in the direction of the apex of the prism. If a prism of, say, 8° has been successfully slipped before one of the child's

eyes with the apex towards the nose, the eye will, in many cases, make a slight inward rotation, showing that the child has some sort of binocular vision. In some cases, however, while the naked eye continues to be steadily directed towards the object, the eye behind the prism makes no inward rotation. The vision of this eye is probably suppressed. These resemble certain cases of occasional squint, in which binocular vision is present when the eyes are "straight," but suppression of the vision of the deviating eye takes place when the squint is manifest.

After the end of the first year, a child who allows one to make the experiment with the prism apex inwards will almost always turn in the eye in order to blend the images.

When the experiment is tried with the prism apex up or down, it is, of course, not possible for the eye to make an independent vertical movement in order to blend the images. I tried this vertical prism with some of the more tractable infants, aged twelve to eighteen months, who had readily made the compensating inward movement when tried with the prism apex in. Some of them showed their disapproval of the vertical prism by screwing up the eyes and twisting the head. Others made up-and-down conjugate movements of both eyes, sometimes directing their attention to the upper image and sometimes to the lower.

To recapitulate—the vision of each eye sepa-

rately, the preponderance of the macular region, and the conjugation of the two eyes in vertical movements, the human infant has fairly well developed at birth. The conjugation of the eyes for horizontal movements (intended to subserve the function of binocular vision) is perfected within the first few months of life. Between five and six months one finds the first certain evidence of a "desire for binocular vision," though probably a certain degree of binocular vision is present at a much earlier period. At first, if any obstacle be interposed, it is a question whether an effort shall be made to overcome it, or whether the newly acquired art shall be abandoned and the vision of one eye temporarily suppressed. Towards the end of the first year the eyes will make a considerable effort in the interests of binocular vision. If the obstacle prove insuperable the child suffers from diplopia, being no longer able to suppress the vision of one eye.

The results of fusion training in the case of squinters would seem to show that the fusion faculty normally reaches its full development before the end of the sixth year.

CHAPTER III

CONVERGENT SQUINT

In most of the text-books squint is defined somewhat as follows: "Squint consists in a deviation of the visual axis of one of the eyes from the correct position of fixation." The authors have mistaken a single symptom for the whole disease. One might as well describe Pott's disease as "a deviation of the spine from its normal shape."

Two essential conditions are present in every case of comitant convergent squint:—

(1) An abnormal convergence of the visual axes.

(2) A defect of the fusion faculty.

Other conditions may also be found:—

(3) The vision of the eye which is not being used for fixation is almost invariably suppressed.

(4) There is, in rather rare instances, more or less congenital amblyopia.

(5) There is very often acquired amblyopia in the deviating eye, as the result of neglect or inefficient treatment.

(6) There is usually a refractive error, commonly hypermetropia and hypermetropic astigmatism.

In a healthy person, with a normal fusion faculty, the "desire for binocular vision" causes the two eyes to be directed steadily to the same object. But when the "desire for binocular vision" is absent there is no special reason for this perfect accord between the movements of the two eyes, so that any slight cause may then upset the equilibrium of the convergence centre, and so cause the visual axes to assume permanently faulty relative directions (see chapter iv.).

Then, when the patient is looking at a distant object, instead of the visual axes being parallel they are convergent. But, in order to see the object distinctly, the patient must look directly at it with one or other eye. He will naturally choose the eye which has the smaller refractive error. He cannot overcome the abnormal convergence, neither can he move one eye without the other. He therefore makes a conjugate lateral movement of both eyes until he has brought the better eye into the required position, the other eye turning still more in towards the nose. So that the better eye becomes "straight," and the worse eye manifests the convergence of both. The eye which is used for vision is called the fixing eye; the other eye is called the squinting or deviating eye.

If a patient fixes, say, with the right eye and turns in the left, he is said to have convergent squint of the left eye. This is a useful convention, but it must be remembered that it does not accurately

describe the condition, as, of course, convergent squint really concerns both eyes and certain cerebral functions as well.

In a typical case of convergent squint the separate movements of each eye are perfect. When one eye is covered, the other eye can move upwards (superversion), downwards (subversion), inwards (adversion), and nearly always outwards[1] (abversion) to the normal extent.

The conjugate movements of the two eyes are perfect. When the fixing eye moves in any direction, the deviating eye also moves through exactly the same angle. When the (previously) fixing eye is screened, and the patient fixes with the (previously) squinting eye, the screened eye manifests a deviation exactly equal to that formerly exhibited by the other eye.[2] In other words, the squint is comitant.

The association between accommodation (dynamic refraction) and dynamic convergence is perfect. When the fixing eye, after looking at a distant object, suddenly accommodates for a near

[1] In 1,523 cases of convergent squint, in which I noted the power of abversion of each eye separately, I found it perfect in 81 per cent. The remaining 19 per cent., in which abversion was deficient, were mostly cases of long standing. The defect of abversion was less uncommon the longer the duration of the deviation.

[2] In a case of uncorrected anisometropia the patient may accommodate to a different degree, according to the eye he uses. This may make the squint appear to be not strictly comitant.

object, the squinting eye rolls still further inwards, a dynamic convergence being superadded to the abnormal static convergence. This dynamic convergence is proportionate to the extra effort of accommodation involved in looking at the near object.

In fact, there is no motor defect of any kind in a typical case of convergent squint, but the primary position from which these movements start is a "cross-eyed" position instead of parallelism of the visual axes.

Convergent squint presents certain clinical varieties. These may be classified as follows :—

(1) Occasional squint, of which there are two classes : (*a*) Premonitory occasional. (*b*) True occasional.

(2) Constant unilateral squint.

(3) Alternating squint, of which there are two distinct classes : (*a*) Accidentally alternating squint. (*b*) Essentially alternating squint.

(1) *Occasional squint.*—A patient is said to have an occasional squint if the eyes are only occasionally seen to deviate from their normal relative directions. When no deviation is present, the patient usually has the first grade of binocular vision. When he squints, he, in the majority of cases, has no diplopia. The deviation of an occasional squint is in some cases only seen for a few seconds in the day, in others the eyes are almost as often "crossed" as not. The deviation in different

cases may manifest itself under various conditions, *e.g.*, in near vision, on looking down, under the influence of any strong emotion such as fear or anger, when the patient is tired, etc. Most frequently no immediate exciting cause can be assigned. Occasional squints are sometimes called *periodic*. I have notes of a few cases which may properly be called periodic squints, in which the deviation has appeared every alternate day as regularly as a tertian ague.

(*a*) Premonitory occasional squints are simply the precursors of constant squints. They usually become constant after about two or three months.

(*b*) True occasional squints are much less common than the premonitory variety. A true occasional squint may gradually cease to show itself as the fusion sense develops, or perhaps optical correction of any refractive error may relieve the condition, or it may maintain its character unchanged throughout life. Many of these last are not squints at all, in the strict sense of the word, but are examples of esophoria, the pathology and treatment of which are described in chapter xi.

(2) In a case of *constant unilateral* squint the deviation is constantly present, though the angle may vary ; and it is always manifested by the same eye, when both eyes are uncovered. When the "fixing eye" is screened, it turns in towards the nose, and the "squinting eye," instead, is directed to the object which engages the attention (unless

this eye has lost the power of central fixation). When the screen is removed, the fixing eye immediately recovers itself, and the squinting eye again turns in.

(3) *Alternating squint.*—When a squinter fixes with either eye indifferently, without covering the other, the squint is said to alternate. Of all constant squints, about 85 per cent. are unilateral and 15 per cent. are alternating. These alternating cases arrange themselves into two distinct classes—

(*a*) Squints which accidentally alternate, because the refraction is about the same in each eye, do not essentially differ from unilateral squints. In fact, if these cases are untreated, usually one eye gradually comes to be used exclusively for fixation, and the squint becomes unilateral.

(*b*) In a case of essentially alternating squint there is usually little or no refractive error, and the visual acuity of each eye separately is, as a rule, perfect. But these patients have a congenital total inability to acquire fusion. Alternating squinters suffer much less than unilateral squinters from neglect, because each eye is used in turn, so that the vision does not deteriorate. If the wearing of glasses does not cause the abnormal convergence to disappear (it seldom does), an accurately performed operation will remove the deformity. The total absence of the fusion sense renders fusion training of no avail in a case of essentially alternating squint.

Suppression of the vision of the deviating eye.—In a case of convergent squint, as the two eyes are not directed towards the same object, it might be thought that everything would be seen double. This is not so, however, save in the exceptional instances referred to below. The visual acuity of the deviating eye may be perfect, but the picture formed in this eye is mentally ignored or "suppressed," the attention being directed solely to that formed in the fixing eye. This "suppression" is not a voluntary act. The inability to receive impressions from both eyes simultaneously is due to the defect of the fusion faculty, which was the essential factor in allowing the squint to occur in the first instance.

This suppression, however, does not always extend over the whole field of vision of the squinting eye. If the deviation be not of high degree, there is a small part of the temporal side of the field of vision of the squinting eye which lies beyond the limit of the field of the fixing eye. In this small area moving objects are perceived by the squinting eye, but, as a rule, are not accurately located. Thus, in an old case of convergent squint, the deviating eye may have become almost totally blind except in the extreme nasal side of the retina (corresponding to the extreme temporal side of its field of vision), the only part which is ever exercised. If the fixing eye in such a case be covered, and the patient be told to look at a

light with the deviating eye, this eye will be seen to roll still further inwards, in order to receive the image of the light on the nasal side of the periphery of the retina.

Occasionally one meets with a squinter who has diplopia of a faint, unobtrusive kind. Squinters never complain of diplopia as a trouble, but one now and then meets with a child who, on being carefully questioned, admits seeing a second image. If the white handle of an ophthalmoscope be held up, such a child will point to the real handle and also to "the sham one," showing that he has really a faint homonymous diplopia, though, in the great majority of cases, he has never mentioned it before. In such a case he evidently has some trace of a fusion faculty, but not sufficiently well developed to prevent the occurrence of a deviation.

Diplopia artificially produced.—In a case of unilateral or accidentally alternating squint, if the deviating eye be not too blind, diplopia may usually be induced by artificial means. For instance, let the patient's attention be directed to a candle flame. Place in a spectacle frame a red glass before one eye and a green glass before the other. The images of the candle flame, formed in the two eyes, being thus differently coloured, the patient is often enabled to perceive them both simultaneously.

Nature of diplopia.—The popular idea is that a squinter sees the object which lies in the axis of the deviating eye as well as that which lies in

the axis of the "straight" eye, so that he can keep an eye on two places at once. Even in the case of the very few squinters who are able to see double spontaneously, or of those who are enabled to do so by artificial means, this view is not correct. A squinter who suffers from diplopia sees, with his deviating eye, a faint, eccentrically placed image of the object to which the fixing eye is directed, and suppresses the image of the object which lies in the axis of the deviating eye. In other words, he does not see two different objects, but sees two images of the same object.

The direction in space in which this second image is mentally projected is peculiar.

Now, in a case of paralysis of, say, the right external rectus muscle, if the right eye deviates inwards to the extent of 20°, everything seen with this eye will appear to be exactly 20° more to the right than it really is; in other words there is homonymous diplopia of 20°. If an eye turns out to the extent of 20° owing to paralysis, there is crossed diplopia of exactly 20°. The mind entirely ignores the faulty position of the eye, and projects everything rigidly as though the eye were straight. In a case of convergent squint, on the other hand, if there is diplopia this does not necessarily correspond in degree with the angle of the deviation of the eye. The eccentrically formed image is, in any case, very faint. Even the most intelligent patient usually is unable to describe its position

exactly, as the angle of the diplopia seems to vary without any corresponding variation in the angle of the squint. In most cases the false image is placed about half-way between the true position of the object and the position which would correspond to the angle of the squint. It would seem as though the mind, being informed by the straight eye of the true position of the object, were continually trying to reconcile this knowledge with the impressions produced by the eccentrically placed false image.

The amblyopia of convergent squint.—When a patient first comes under observation after having suffered from unilateral squint for a considerable time, one usually finds that the deviating eye is more or less blind, so blind sometimes that fingers can scarcely be counted close to the face. This amblyopia is sometimes, to a certain extent, congenital. But by far the greater part of it is due to a gradual loss of function in an eye which is never used. It might have been prevented. This is plainly seen by comparing the vision in my cases which came under treatment soon after the first appearance of the squint, with the vision in the cases which I saw for the first time only after years of neglect or inefficient treatment (see pages 76 and 77).

The power of central fixation in the deviating eye.—In a case of unilateral convergent squint, if the fixing eye be covered the vision of the (previously) deviating eye temporarily ceases to be

suppressed. In a fairly recent case, this eye is then directed so as to receive, upon its macula lutea, the image of the object looked at. But if the case be long neglected, this sensitive central region of the retina suffers much more from disuse than the paracentral zone, while the peripheral region suffers very little, if at all. As the blindness progresses in this disused eye, a stage is at length reached when the visual acuity of the central region falls below that of the paracentral zone, and later, even below that of the periphery of the retina. If the fixing eye be now covered, the deviating eye is not directed so as to receive upon its macula the image of the object which engages the attention, because the macula has ceased to be the most sensitive part of the retina. This eye then wanders, without remaining steadily in any definite position (*lost fixation*). Or it may fix with some part of the paracentral region, or roll still further in towards the nose so as to present the extreme nasal periphery of the retina for the purpose (*false fixation*).

False macula.—False fixation is, unfortunately, exceedingly common in neglected cases of unilateral squint. But the variety known as false macula is rare. In an old case of squint, in which the angle of the deviation has remained exactly the same for several years, and in which the suppression of the vision of the deviating eye is not profound, the mind sometimes learns to make full allowance for

the faulty position of this eye. So that the eccentric image, formed in the deviating eye, is mentally projected to the same spot as the true macular image, formed in the normally directed eye, and is blended with it. This false macula is merely a small area which has escaped the loss of function which has overtaken the surrounding part of the retina. The visual acuity of a false macula is never greater than the normal visual acuity of the region in which it is situated. I have rarely found it equal to $\frac{3}{60}$; never greater. Many of these patients can pass Hering's drop test. If an eye, with false macula in a position of convergence, be put "straight" by operation, crossed diplopia is produced. This usually passes off within a few days, but occasionally it persists for many months.

Apparent vertical deviation.—On applying the mirror test in a case of convergent squint, one not infrequently notices a slight upward deviation also: then, on covering the originally fixing eye, and causing the originally deviating eye to fix, one sees that the originally fixing eye also turns *up* as well as in. If the wearing of glasses causes the convergence to diminish, the vertical deviation will also diminish proportionately. In the great majority of cases the vertical deviation increases when the gaze is directed towards the side of the fixing eye and diminishes or disappears when the gaze is directed away from the side of the fixing eye.

In some cases of divergent squint, the divergent eye turns a little down also, no matter which eye may be fixing.

The explanation is that in these cases the planes of lateral rotation of the two eyes, instead of being horizontal, are tilted down and out towards each temple. Then, when the fixing eye is moved horizontally, the deviating eye, in making a corresponding conjugate movement uncontrolled by the fusion sense, moves in this tilted plane. Take, for example, a case of convergent squint 20°, exhibiting this double upward deviation. Really each eye is convergent 10°, but the eyes have made a conjugate movement of 10° towards the side of the fixing eye so as to bring this eye "straight" and double the apparent convergence of the deviating eye. The fixing eye has thus travelled out and down in its plane, and the deviating eye has travelled in and up. A further conjugate movement towards the side of the fixing eye will increase the difference in vertical height. A conjugate movement away from the side of the fixing eye, by bringing the eyes to corresponding points in their respective planes of rotation, will cause the vertical difference to lessen or disappear.

True vertical deviation.—In some cases of squint there is a true vertical deviation, one eye turning up when it becomes the deviating eye, and the other eye turning down when it, in turn, is made to deviate.

The distinction between true and apparent vertical deviation is of great importance in view of the treatment.

Spurious squint of infants.—During the first few months of life, before the fusion faculty has made much progress in development, it frequently happens that the eyes converge for a few seconds or a minute at a time, in response to some gastric or other disturbance. Or, as nurses are wont to express it, "Babies squint when they have the wind." This is of no importance. Sometimes, too, a child, whose fusion faculty is developing quite normally in other respects, acquires this faculty rather later than usual, just as a perfectly healthy, intelligent child may be late in learning to talk. One may then see one or other eye turn in, occasionally, for a minute or two at a time, even when the child is old enough to walk. Later, if the fusion faculty develops normally, it will so control the movements of the eyes as to prevent any recurrence of the deviation.

But if the child "crosses his eyes" for many minutes at a time, or if one eye converges while the other steadily fixes some object, or, more especially, if the deviation is always manifested by the same eye, the case is probably one of true squint and demands investigation without delay. And, under any circumstances, it is safer to investigate any case in which a child is seen to cross his eyes occasionally, rather than wait until what

may have been a premonitory occasional squint becomes a constant squint.

"*Growing out of a squint.*"—With the advent of puberty, the angle of a convergent squint often tends to become somewhat less without any treatment. In rather rare instances the eyes become straight, or nearly so, and the patient is said to have "grown out of the squint." But, in the case of a unilateral squint, the squinting eye has nearly always by this time become very blind from disuse.

The belief in the spontaneous cure of squint is very widespread among the general public. This may be due partly to the fact that the deformity of squint does, in a few cases, disappear spontaneously as just mentioned, and partly to the fact that an occasional squint is seen at one time and not at another, but chiefly, I think, to the fact that the spurious squint of infants ceases to manifest itself when the fusion sense develops.

But, unfortunately, this superstition is not entirely confined to the laity. I have often had a child brought to me with the squinting eye nearly blind from neglect, and have been told that the family practitioner was consulted about it years ago and that he advised the parents to "wait to see if the child would grow out of the squint."

General course of an untreated case of constant unilateral convergent squint.—At the first appear-

ance of a deviation, the squinting eye always has the power of central fixation when the fixing eye is covered, and the vision is nearly always good in both eyes. There is rarely some congenital amblyopia. This congenital amblyopia is far less frequent than is generally supposed, and is never responsible for the extreme blindness so often found in old neglected cases of squint.

In an untreated case the vision of the deviating eye, being entirely suppressed, gradually deteriorates from disuse, until, in many cases, central fixation is lost, and the vision reduced to the counting of fingers close to the face. The younger the child the more readily does this amblyopia from disuse occur. So much so that it is commonly believed, even by ophthalmic surgeons,[1] that an eye which begins to squint in early infancy is necessarily very blind, and this blindness is supposed to be congenital. Yet this is not the case. Of the cases which came under my care soon after the first appearance of the deviation I do not find the young squinters especially amblyopic (see chapter v.).

The deviation, in an untreated case of convergent squint, usually increases in degree up to the time of puberty, and afterwards remains nearly stationary. Rarely, however, it tends to become

[1] This was written many years ago. Fortunately for the patients, at the present time there are few ophthalmic surgeons who hold this view.

gradually less, until about forty years of age, when in some cases it may be scarcely noticeable. Occasionally one sees a middle-aged patient with an inconspicuous squint of perhaps two or three degrees, and a nearly blind eye. He usually says he squinted when he was a boy, but gradually grew out of it.

Age at which the deviation first appears.—In my note-books I find the onset-age recorded in 1,017 cases of unilateral convergent squint, and in 178 alternating cases.[1]

The number of *unilateral* cases beginning in each year of life was as follows:—

Before 1 year	134 cases.
Between 1 and 2 years	186 ,,
,, 2 ,, 3 ,, ...	247 ,,
,, 3 ,, 4 ,, ...	189 ,,
,, 4 ,, 5 ,, ...	113 ,,
,, 5 ,, 6 ,, ...	73 ,,
After 6 years	75 ,,

It will be seen that in nearly 75 per cent. of the cases the deviation appeared before the end of the fourth year, and in less than $7\frac{1}{2}$ per cent. its advent was delayed until after the sixth year.

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[1] Up to the year 1902. As the number is amply sufficient to eliminate any element of chance, further pursuit of this enquiry with material which has been subsequently available would have served no useful purpose. This applies to all the statistics in this and the two following chapters.

The *alternating* cases work out as follows :—

Before	1	year	...	...	...	61	cases.
Between	1	and	2	years	...	34	,,
,,	2	,,	3	,,	...	23	,,
,,	3	,,	4	,,	...	29	,,
,,	4	,,	5	,,	...	11	,,
,,	5	,,	6	,,	...	6	,,
After	6	years		...	...	14	,,

In more than 53 per cent. of these alternating cases the deviation was seen before the end of the second year. This high proportion is due to the fact that the essentially alternating squints appear in early infancy.

Refractive error in cases of convergent squint.—In infancy and early childhood hypermetropia is the normal refractive condition, myopia or even emmetropia being then very uncommon. In the absence of the controlling influence of the fusion sense, the state of the refraction is the main factor in determining whether the eyes shall deviate inwards or outwards (see chapter iv.). It is not surprising, therefore, that convergent squinters are nearly always hypermetropic, and very frequently suffer from hypermetropic astigmatism also.

I have notes of 1,636 cases of convergent squint which are available for the present enquiry.

In twenty-three of these cases, or about 1½ per cent., both eyes were myopic. I have not included these in the tables.

I have arranged the 1,384 cases of unilateral

Table I.

One thousand three hundred and eighty-four cases of unilateral convergent squint, arranged in groups according to the degree of refractive error in the lowest meridian of the fixing eye.

Group. Refractive Error in Lowest Meridian of Fixing Eye.	Number of Cases.	Average Age of Onset.	Average Refractive Error.			
			Fixing Eye.		Deviating Eye.	
			Lowest Meridian.	Highest Meridian.	Lowest Meridian.	Highest Meridian.
		Years. Months.				
Not above + 1 D	83	2 10	0·45	1·36	0·48	1·7
Above + 1 D. Not above + 2 D	142	2 11	1·6	2·5	1·93	2·98
„ + 2 D. „ „ + 3 D	240	3 1	2·7	3·2	2·95	3·74
„ + 3 D. „ „ + 4 D	285	3 0	3·61	4·3	3·9	4·82
„ + 4 D. „ „ + 5 D	292	3 0	4·72	5·4	4·84	5·81
„ + 5 D. „ „ + 6 D	209	2 11	5·64	6·62	5·9	7·05
„ + 6 D. „ „ + 7 D	77	3 0	6·5	7·6	6·92	8·27
„ + 7 D. „ „ + 8 D	31	2 10	7·4	8·1	7·6	8·53
„ + 8 D. „ „ + 9 D	16	3 2	8·5	9·25	9	9·84
„ + 9 D. „ „ + 10 D	9	3 5	9·55	10·33	9·66	10·88

convergent squint in groups, according to the number of dioptres of hypermetropia in the lowest meridian of the fixing eye.

I have calculated the average refractive error in the highest and in the lowest meridian of each eye. In about three-fourths of these cases the age at which the squint was first seen was recorded on the case-sheets. The table shows the number of cases found in each group, and the average onset-age.

The 229 cases of alternating convergent squint are similarly tabulated, except that they are arranged in groups according to the number of dioptres of hypermetropia in the right eye.

Astigmatism in unilateral convergent squint.—In the 1,384 cases I found the proportion of astigmatic to non-astigmatic eyes to be as follows :—

	Fixing Eyes.	Deviating Eyes.
No astigmatism ...	561	401
Astigmatism	823	983

But a very large proportion of people whose eyes are, for all practical purposes, normal, have at least half a dioptre of astigmatism. It would only be misleading to expect the eyes of squinters to conform to a higher standard than those of normal-sighted people. If we disregard astigmatism which does not exceed 0·5 D, we get the following results :—

	Fixing Eyes.	Deviating Eyes.
Astigmatism not over 0·5 D	836	628
Astigmatism over 0·5 D	547	756

Table II.

Two hundred and twenty-nine cases of alternating convergent squint, arranged in groups according to the degree of refractive error in the lowest meridian of the right eye.

Group. Refractive Error in Lowest Meridian of Right Eye.	Number of Cases.	Average Age of Onset.	Average Refractive Error.			
			Right Eye.		Left Eye.	
			Lowest Meridian.	Highest Meridian.	Lowest Meridian.	Highest Meridian.
		Years. Months.				
Not above + 1 D	33	0 11	0·6	0·81	0·63	0·79
Above + 1 D. Not above + 2 D	34	1 7	1·5	1·9	1·45	1·95
„ + 2 D. „ „ + 3 D	40	2 9	2·61	3·1	2·7	3·15
„ + 3 D. „ „ + 4 D	41	2 10	3·58	4·25	3·4	4·16
„ + 4 D. „ „ + 5 D	36	2 10	4·7	5·41	4·6	5·45
„ + 5 D. „ „ + 6 D	25	2 8	5·65	6·14	5·71	6·18
„ + 6 D. „ „ + 7 D	9	2 11	6·66	7·27	6·77	7·13
„ + 7 D. „ „ + 8 D	5	3 2	7·65	8·4	7·4	8·6
„ + 8 D. „ „ + 9 D	4	4 0	8·75	9·5	8·75	9·37
„ + 9 D. „ „ + 10 D	2	2 6	9·75	10·5	9·75	10·75

On comparing the degree of refractive error in the two eyes in each of the 1,384 unilateral cases we find :—

Isometropia	427 cases.
Anisometropia	957 ,,

Or, disregarding differences which do not exceed 0·5 D :—

Anisometropia not exceeding 0·5 D ...	663 cases.
Anisometropia exceeding 0·5 D ...	721 ,,

These *alternating cases* show a much lower percentage of astigmatic eyes than the unilateral cases :—

	Right Eye.	Left Eye.
No astigmatism	114	112
Astigmatism	115	117

If we disregard astigmatism which does not exceed 0·5 D, we find :—

	Right Eye.	Left Eye.
Astigmatism not over 0·5 D	140	141
Astigmatism exceeding 0·5 D	89	88

A comparison of the refraction of the two eyes in each of the 229 alternating cases shows :—

Isometropia	131 cases.
Anisometropia	98 ,,

Or, disregarding differences which do not exceed 0·5 D :—

Anisometropia not exceeding 0·5 D	185 cases.
Anisometropia exceeding 0·5 D ...	44 ,,

The relative frequency of squint was the subject of a careful enquiry which Mr. R. E. Hanson,

who was at that time oculist to the Education Department of the London County Council, was kind enough to undertake at my request. During 1901 and 1902, 10,239 school-children were examined in the Marylebone and the Tower Hamlets districts. 253 of these children exhibited a constant squint. In 231 of the cases the squint was convergent, and in 22 cases divergent. The test used was the mirror test described on page 80.

CHAPTER IV

THE ÆTIOLOGY OF CONVERGENT SQUINT

MANY curious suggestions have, in former times, been made as to the cause of squint, such as "an evil disposition," naughtiness, imitating other members of the family who squint, the habit of turning the eye to look at a curl or ribbon on one side of the face, etc. The first definite theory, which was almost universally accepted, attributed convergent squint to a shortening of the internal recti. The natural corollary of this was that the affection might be cured by dividing these muscles or their tendons. The theory and its practical application seemed so plausible and simple that an age of indiscriminate muscle-cutting ensued. When the disastrous results of this practice were beginning to be recognised, Donders published his great work, and his "accommodation theory" of the cause of squint immediately came into vogue.

It will be well to examine these two theories in detail.

Muscle theory.—Many writers on convergent squint have attributed the anomaly to an undue shortness or tightness of the internal recti, to a

faulty insertion of the tendons of these muscles, or to a paresis of the external recti. To an observer who sees in a case of convergent squint only its most obvious symptom, viz., the abnormal convergence, it may seem reasonable to attribute this deformity to a defect of the muscles which move the eyes. A little investigation ought to convince anyone of the falsity of this view.

Of 1,523 cases of convergent squint in which I investigated this point, I found the power of abversion (outward rotation) of each eye tested separately to be perfect in 81 per cent.; in the remaining 19 per cent. the power of abversion was sub-normal. This alone would seem sufficient to demonstrate that, in 81 per cent. of convergent squints at least, there is no undue shortness of the internal recti or paresis of the external recti muscles. In the 19 per cent. the deviation had, in most cases, lasted several years, and the average degree of the defect of abversion was roughly proportionate to the duration of the deviation. The defect of abversion in most of the 19 per cent. of cases would therefore appear to be due to *secondary* changes in the muscles and fasciæ, the result of the long continuance of the deviation, not its cause. One wonders that these secondary changes are not more marked.

It is a matter of common observation that a convergent squint very frequently disappears, or the eyes even diverge, during general anæsthesia.

Occasionally a very high degree of convergence disappears when the accommodation is paralysed by atropine. It always reappears when the effect of the atropine has passed off.

Of cases of convergent squint which are treated with glasses alone, in a considerable proportion[1] the eyes eventually become approximately "straight," and remain so as long as the glasses are worn.

These facts scarcely seem compatible with a muscular origin of squint, except perhaps in a very small proportion of cases.

Donders' theory.—When a person with normal emmetropic eyes looks at an infinitely distant object, such as a star, the visual axes are parallel, and the static refraction of each eye is sufficient to focus the image of the object on its retina. If the person now looks at an object only a foot away from him he must turn the eyes slightly inwards (convergence), in order that both visual axes may meet at the object. At the same time each eye

[1] I have no statistics of my own showing the proportion of "cures" effected by glasses alone, because I do not rely solely upon optical correction and operation. Of ninety-four cases of unilateral convergent squint quoted by Holthouse, the deviation was gradually overcome, and the eyes remained "straight" so long as glasses were worn in twenty-nine cases, or 30·8 per cent. Lang and Barrett (*R.L.O.H. Reports,* vol. xii.) had thirty-seven cures by glasses out of 102 cases, or 36·3 per cent. But in this series all cases in which less than five degrees deviation remained were considered cured, and the enquiry was only continued during a short time.

must be focussed for near vision (accommodation), in order that the object may be seen distinctly. These two acts, accommodation and convergence, being always performed together, have become "associated" by hereditary habit, so that it is difficult to converge without accommodating or to accommodate without converging.

Hypermetropic eyes in a state of rest are out of focus even for distant objects, and still more so for near objects. A hypermetrope, therefore, in order to see distinctly, must accommodate in distant vision to a degree corresponding to the amount of his hypermetropia. In near vision he must accommodate both for the hypermetropia and for the nearness of the object. There is a *tendency* for a proportionate abnormal convergence to be associated with this abnormal effort of accommodation. Donders considered this tendency to be the cause of convergent squint, and he advised optical correction of the hypermetropia with a view to curing the squint.

By this chain of observation and reasoning the great physiologist let the first ray of light into this dark corner of ophthalmology, and gave the first indication for a rational treatment of convergent squint. But he was mistaken in supposing hypermetropia to be the fundamental cause of the malady.

The vast majority of children are hypermetropic. Of these hypermetropes only a small percentage

present themselves at the clinics, whereas nearly all the squinters come at some time or other. Yet, even there, one sees at least a dozen hypermetropes who do not squint for one who does. This cannot be explained on the hypothesis that the severe cases of hypermetropia especially tend to cause squint, because statistics show that this is not the case (see tables i. and ii., chapter iii.).

It is usually stated that moderate degrees of hypermetropia are especially prone to be associated with squint. This has been explained by saying that a child with moderate hypermetropia will accommodate in order to see distinctly. He only manages to make the extra accommodative effort by bringing into play also an associated effort of convergence, so that he sees distinctly with one eye and turns in the other. But, it is said, if the hypermetropia be excessive he will not be able to accommodate sufficiently, so that he gives up the struggle, and neither sees distinctly nor squints.

But this ingenious argument is based on false premises. Moderate degrees of hypermetropia are more common than high degrees, not only in those who squint, but also in those who do not squint. I looked through my out-patient letters at the West Ham Hospital for about eighteen months, and tabulated all the cases of hypermetropia, without squint, in which the refractive error was measured by retinoscopy under atropine. I tabulated the cases according to the degree of hypermetropia, and

worked out the percentage number of eyes in each grade.[1]

The results, in the cases in which the hypermetropia was more than 2 D, were found to correspond very closely with my squint statistics treated in the same way. The proportion of cases with less than 2 D was lower among the non-squinters than among the squinters. Also the average degrees of astigmatism were higher among the non-squinters than among the squinters. This is evidently due to the fact that most of the non-squinters were patients who sought advice on account of headaches or visual defect.

It will thus be seen that the *degree* of the refractive error has very little to do with the question of whether the patient shall or shall not squint in the first instance, though, of course, when the squint is once established, the refractive error becomes a very important factor.

A high degree of hypermetropia has no influence in determining an early onset of squint, as is conclusively shown by the following table. In the unilateral cases I have taken the average of the

[1] In cases in which there was also astigmatism, the mean between the highest and lowest meridian was taken. I have since compared the results of taking the highest meridian only, also the lowest meridian only. The number of cases taken excludes any accidental source of error. A slight defect in this mode of comparison will, of course, suggest itself, but the error is not sufficiently grave to affect the main results.

highest and lowest meridian in the fixing eye, and in the alternating cases I have taken the average of both eyes.

Less than + 2 D	average age of onset	= 2·47 years.[1]	
+ 2 D to + 4 D	,,	,,	= 2·85 ,,
+ 4 D to + 6 D	,,	,,	= 2·92 ,,
More than + 6 D	,,	,,	= 2·96 ,,

A great many children who suffer from convergent squint have no more than the normal degree of hypermetropia, while 1 or 2 per cent. are actually myopic.

It is evident, then, that, though hypermetropia stands in some close ætiological relation to convergent squint, it is not the essential cause of the anomaly.

ÆTIOLOGY OF CONVERGENT SQUINT.

For the sake of clearness, I will first state my proposition, and give the proofs afterwards.

In a case of convergent squint there is, in addition to the most obvious symptom, the deformity, always a defect of the fusion faculty, and there is nearly always a suppression of the vision of the deviating eye.

In the human infant the motor co-ordinations of the eyes are already partially developed at birth. During the first few months of life these serve (in

[1] The average age of onset is low in these squinters, with less than 2 D of hypermetropia, because cases of essentially alternating squint are included in this table.

the absence of any disturbing influence) to maintain *approximately* the normal relative directions of the eyes. Soon the fusion faculty begins to develop. I have found distinct evidence of binocular vision in the sixth month. Normally the development of the fusion faculty is well advanced by the twelfth month, and complete before the end of the sixth year. When the fusion faculty has begun to develop, the instinctive tendency to blend the images formed in the two eyes—the "desire for binocular vision," as it is called—will keep the eyes "straight." When the fusion faculty is fairly well developed, neither hypermetropia, nor anisometropia, nor heterophoria can cause squint. In fact, then, nothing but an actual muscular paralysis can cause an eye to deviate, in which case the resulting diplopia is intolerable. Sometimes, however, owing to a congenital defect, the fusion faculty develops later than it should, or it develops very imperfectly, or it may never develop at all. Then, in this case, there is nothing but the motor co-ordinations to preserve the normal relative directions of the eyes, and anything which disturbs the balance of these co-ordinations will cause a permanent squint. Thus the *essential cause of squint is a defect of the fusion faculty*. In the presence of this fundamental cause the eyes are in a state of unstable equilibrium, ready to squint either inwards or outwards on slight provocation. This provocation may be supplied by :—

(1) *Hypermetropia.*—As already explained, uncorrected hypermetropia causes a *tendency* to abnormal dynamic convergence of the visual axes. In the vast majority of cases of hypermetropia the fusion sense is perfect, so that this tendency is kept in check and the child does not squint. If, however, the fusion sense is deficient, the eyes are free to yield to this tendency, and a convergent squint is established. In the cases in which hypermetropia is the immediate exciting cause, the abnormal convergence is at first entirely dynamic, static convergence being *nil*. The squint is at first occasional—when the child is looking at nothing in particular, relaxing the accommodation, the deviation disappears. Optical correction of the refractive error at this period often cures the deviation. But if nothing is done, the excessive exercise of the function of dynamic convergence causes an abnormal static convergence to appear. So that the visual axes are convergent even when the eyes are completely at rest. At this period optical correction does not cause an immediate disappearance of the deviation. It may gradually do so, perhaps, after the glasses have been worn for a few weeks or months.

In cases in which the fusion sense is present but feeble, it may be strong enough to resist the strain of uncorrected hypermetropia during infancy, but may give way when the child's attention is directed to his first lessons. There is scarcely ever diplopia.

Rarely one meets with a child who, when patiently questioned, admits that he sees a second image. This faint diplopia persists, but is never annoying.

During the first few weeks of life, even in cases of very high hypermetropia, the motor co-ordinations suffice to maintain approximately the normal relative directions of the eyes, until the developing fusion faculty takes control and makes everything safe. Probably the infant does not use his accommodation, except for brief periods, during the first four or five months of life. But, occasionally, hypermetropia may cause a squint before the period at which the child should normally have made useful progress in acquiring the fusion faculty (*e.g.*, Case A, 503, page 155). In such a case, if the deviation is allowed to persist, the natural development of the fusion faculty will, of course, be prevented.

(2) *Anisometropia,* and the rare congenital amblyopia, predispose to squint by making binocular vision more difficult. One occasionally sees a case of squint in which the fixing eye is approximately emmetropic and the deviating eye has perhaps 10 or 15 D of myopia.

(3) *Motor anomalies.*—If the fusion sense be perfect, a want of balance of the motor apparatus of the eyes will cause heterophoria but not squint. But in the absence of the controlling influence of the fusion sense this motor imbalance is free to cause an actual deviation. This is seen when, in

examining a case of heterophoria, fusion is temporarily rendered impossible by means of Maddox rods, coloured glasses, etc.

(4) *Specific fevers,* especially whooping-cough, are often assigned by the parents as the cause of the squint. One usually hears that the child was seen to squint during convalescence, that the squint was, at first, occasional, and that it became constant after a few weeks. These children usually have a good deal of hypermetropia. The mode of origin of these squints is probably as follows: The fusion sense is defective (shown by the absence of diplopia), but the motor co-ordination, or some slight degree of fusion sense, has hitherto sufficed to prevent the occurrence of a deviation. During convalescence the child is given picture-books. Owing to the muscular enfeeblement caused by his illness, he is unable to accommodate so well as formerly. The excessive effort of accommodation upsets the unstable equilibrium. At first the abnormal convergence is dynamic only, and disappears when the child relaxes his accommodation. A pair of spectacles at this period often brings about a cure. But if the opportunity be lost a static convergence appears, and what was merely a premonitory occasional squint becomes constant.

As a contrast to these cases it occasionally happens that a child, whose fusion faculty is perfect, suffers from a paresis of an external ocular muscle after diphtheria. The child complains of persistent and annoying diplopia until the muscle recovers its function. I

recently had the opportunity of watching such a case. The patient, a little girl, aged five years, had paresis of the right external rectus muscle after diphtheria. She volunteered the statement that she saw "two nurses" and that "things looked funny." She was uncertain in her movements in running about, and often made a false shot in picking up a ball from the floor. When I gave her a picture-book to look at, she covered the affected eye with her hand. When I saw her a few days later, she kept her head constantly turned to the right, evidently to enable her to blend the images. The case completely recovered in about seven weeks.

(5) *Violent mental disturbance,* caused by severe fright, "convulsion-fits," etc., may, in the absence of the fusion faculty, upset the equilibrium of the convergence centre. The convergence is static, it appears immediately, is constant from the first, and is usually alternating. Refractive error is not an important feature.

Injury during birth.—Now and then one sees a case in which the power of abversion of an eye has been absent since earliest infancy, probably owing to injury to the sixth cranial nerve during birth. In most of these cases the position of the eye prevents the natural development of the fusion sense. If no precautions are taken the eye may become extremely amblyopic.

Occlusion.—If, for any reason, an eye of a very young child is kept tied up for a long time it may become so amblyopic as to render fusion impossible. Squint may then result.

Hereditary influence is a marked feature in any series of cases of convergent squint. In 1,373 cases of squint in which I was able to get probably reliable information there was a history of squint in parent, grandparent, brother, or sister of the patient in no less than 711, or 51·78 per cent.

Proof that the Essential Cause of Squint is a Defect of the Fusion Faculty.

If a pair of object-slides such as fig. 17 be put in the amblyoscope, a person with a normal pair of eyes will be able to blend the two imperfect images into one complete picture. If the angle of the instrument be varied the eyes will converge, or (to a certain extent) diverge also, in order to follow the objects and maintain fusion.

Now take a young patient with an ordinary unilateral convergent squint, and good vision in each eye. Adjust the amblyoscope to suit the angle of his deviation. He will only see with the fixing eye, the vision of the squinting eye being suppressed. If the suppression be now overcome by the method described in chapter viii., he will see the two imperfect images simultaneously. After a little practice, a position can usually be found in which the two imperfect images overlap, so that the patient sees them blended into one complete picture. But they are blended only in that one position. If the angle of the instrument be again

altered, the images at once separate—no effort can be made to maintain fusion. This shows that, though the fusion faculty is not quite absent, it is exceedingly ill-developed. The results obtained in many of these cases by training the fusion faculty at an early age strongly support the view that the defect of this faculty was the fundamental factor in permitting the deviation to occur.

Consider next a typical case of essentially alternating squint. The visual acuity of each eye is perfect; there is no important refractive error; the movements of each eye separately are perfect. Why then does such a pair of eyes squint? The muscle theory cannot explain it, because there is no motor defect of either eye. Hypermetropia cannot be the cause, because there is little or no refractive error; moreover, the wearing of correcting glasses usually has no effect upon the deviation. No theory hitherto put forward has satisfactorily explained these cases. But if the condition of the fusion faculty be examined the mystery is cleared up at once. Let a patient with an essentially alternating squint look at a pair of object-slides such as fig. 17 in the amblyoscope. When the objects are very far apart, he may be able to see both. But when they are made to approach each other, he loses sight of one of them. No amount of practice will ever enable an essentially alternating squinter to see the two devices simultaneously when they are close together, much less to blend them.

There is a congenital total absence of the fusion faculty.

The great frequency with which squint occurs in more than one member of a family has enabled me to obtain still more direct evidence. When I have had a child under treatment for convergent squint, I have, in very many instances, induced the mother to bring me also for examination a younger brother or sister of the patient, who had not hitherto squinted. I examined the fusion sense of all except the most intractable of these brothers and sisters of squinters with the amblyoscope. I have been able to follow the subsequent history of 157 of these children. In 106 cases I found the fusion faculty well developed. *Not one of these has subsequently squinted.* Of thirty-seven cases in which the note was "doubtful," six have since squinted. Of fourteen cases in which my note said "fusion faculty very deficient," eight have since developed a constant squint, and another child is said to cross her eyes occasionally.

CHAPTER V

AMBLYOPIA, CONGENITAL AND ACQUIRED

THE amblyopia discussed in this chapter is a partial blindness of an eye in which the most careful examination of the fundus and media reveals nothing sufficient to account for the defect. This amblyopia persists after accurate optical correction of any refractive error which may be present. It may be either congenital or acquired.

It will be well to consider congenital amblyopia and the acquired form separately, before discussing the amblyopia so often found in cases of convergent squint.

Congenital amblyopia, apart from squint, is very seldom met with. One should not accept a case as being one of congenital amblyopia, unless careful questioning of intelligent and observant parents makes it certain that the patient has never squinted as a child.

In the course of examining the refraction of many thousands of patients who have never squinted, up to the end of 1902 I only met with twenty-three cases of amblyopia, of $\frac{6}{18}$ or higher, which I felt justified in regarding as congenital. These cases are described in detail in the Appendix (p. 232).

Other similar cases have since been investigated. They have certain peculiarities in common. The fundus and media are normal in appearance. The fields of vision, both for white and colours, are full. There is no scotoma. Central colour perception is normal. The peripheral form vision, up to within 20° of the fixation point, is normal.[1] So that the defect would seem to consist in a want of due preponderance of the macular region, and not in a general lowering of the sensibility of the visual apparatus. In no case was the vision of the amblyopic eye less than $\frac{6}{60}$. But the most remarkable feature of these cases is that the defect is confined to one eye, which almost invariably has a high degree of compound hypermetropic astigmatism, while the other eye has normal vision, and either normal refraction or hypermetropia without any notable degree of astigmatism. In many of the cases the fusion faculty was examined with the amblyoscope. It was found to be well developed.

[1] For the purpose of testing the peripheral form vision, I use three metal screens about two inches square, each showing a white **O** on a black ground. The letters on the three screens are of different sizes. By a simple mechanical device the **O** is readily converted into a **C**. The screens are successively attached to the object-carrier of a perimeter. The investigation is proceeded with just as in mapping out the fields of vision, the patient being required each time to say whether the letter is **O** or **C**. In this way three zones are mapped out on the chart. This method, though very valuable for purposes of scientific investigation, is too tedious for ordinary clinical use.

In some, at least, of these cases in which I formerly regarded the amblyopia as congenital, it may really have been acquired through the patient's confining his attention to the sharp image, even though this is fused with the blurred image of the astigmatic eye. The fact that the amblyopia is always confined to one eye lends support to this view. The possibility of confining one's attention to the image formed in one eye, and yet, at the same time, exercising binocular vision, is easily demonstrated. Let one imitate the refractive condition of one of these patients by wearing a high cylindrical lens before one of one's own eyes, then read the test types with both eyes open. By confining the attention to the naked eye, one reads $\frac{6}{6}$. In reading a book one soon learns to disregard entirely the blurred image seen through the cylinder. At the same time, it is easy to convince oneself that one is exercising binocular vision—prism, base in, produces diplopia; prism, base out, causes convergence of the visual axes in order to blend images; four-dot test, etc.

Wishing to avoid propounding any theory not amply supported by facts, I have, in former editions, put forward this view merely as a tentative suggestion. Further investigation of cases has now fully convinced me of its truth.

There is another small group of cases in which the amblyopia may almost certainly be regarded as congenital. Table iii., p. 76, includes only cases of squint in which my treatment was begun soon after the first appearance of the deviation and was carried out thoroughly, so that there was scarcely a possibility of any acquired amblyopia. It will be seen that the vast majority of these patients have, with optical correction, perfect vision in each eye.

In seventeen cases out of the 193 the vision of the squinting eye was $\frac{6}{9}$ or $\frac{6}{12}$. In nine cases it was $\frac{6}{18}$ or $\frac{6}{24}$. In two cases it was $\frac{6}{36}$ or $\frac{6}{60}$. In no case was the vision lower than $\frac{6}{60}$, and in no case was the power of central fixation absent. On referring to my case-books, I find that, in the more amblyopic cases, the defective eye nearly always had a high degree of compound hypermetropic astigmatism, while the other eye had perfect vision and nearly always simple hypermetropia without any notable astigmatism. These cases are described in the Appendix.

To sum up, congenital amblyopia up to $\frac{6}{12}$ is uncommon; between $\frac{6}{18}$ and $\frac{6}{60}$ it is rare and its origin is open to doubt; I have never seen congenital amblyopia of higher degree than $\frac{6}{60}$.

Amblyopia acquired from disuse (amblyopia ex anopsia). In a case of convergent squint, even though the vision of each eye separately be perfect, the patient will, when both eyes are open, only see with the "straight" eye. The impressions received by the deviating eye are mentally "suppressed." In the case of a young child with a constant unilateral squint, the result of this disuse of the deviating eye is that its visual acuity gradually deteriorates. This deterioration from disuse is the more rapid the younger the child, so much so that it is commonly believed that an eye which squints in infancy is necessarily very blind. This is not so, as table iii. clearly shows. A child with good vision

in each eye, who develops a constant unilateral squint at the age of six or eight months, will, in the absence of proper treatment, become rapidly blind in the squinting eye. This loss of vision in the infant's deviating eye is so rapid that the power of central fixation is often lost within eight or ten weeks. In an eye which begins to squint constantly at the age of, say, eighteen months, the progress of the blindness is rapid, but much less so than in a younger child. At least five or six months usually elapse before the eye loses the power of central fixation. An eye which begins to deviate constantly at the age of three years seldom quite loses the power of central fixation in less than a year. I have never seen central fixation lost in a case in which the squint had first appeared after six years of age. After six years of age amblyopia ex anopsia seldom takes place to any great extent. Acquired amblyopia is a true loss of vision, not a failure of the function to develop, as is shown by cases quoted below.

I have notes of several cases in which ignorant or careless parents have accidentally performed upon their children most instructive (but disastrous) experiments bearing upon this subject. Here are brief notes of a few of the more striking examples.

CASE A, 77.[1]—On November 14, 1895, I saw, at West Ham, a girl, aged 2 years 7 months. She had

[1] The letters and numbers are the index marks of the notes in my squint case books. In the case notes I have

constant convergent squint R. E. 22°. The mother said that the child had squinted about a month. The squint was nearly alternating. After shading L. E. for a moment, she would fix with R. E., and continue to do so for a minute or two after removing the shade. Two hours after putting a drop of atropine into the L. E. she was seen to squint always with the L. E. and fix with the R. E.

November 21, 1895.—Retinoscopy under atropine, each eye + 3·75 D sph. I ordered + 3 D sph. for constant wear; and a drop of liquor atropinæ to be put into the L. E. *only* every morning. Child to be seen again in a month.

August 22, 1901.—The child is brought to me at the West Ham Hospital. I have not seen her for nearly six years. The mother has no recollection of having used the drops for the L. E. She says the child wore the glasses for about a year, then lost them. No treatment since. Child is now aged 8 years 4 months. She has convergent squint R. E. 17°. The fixation of the R. E. is lost. Vision of the R. E. is reduced to the counting of fingers at 5 feet.

CASE D, 527. *October* 16, 1900.—Boy, aged 13 months, brought to Moorfields. The mother said he had "squinted the last few weeks." Child has convergent squint R. E. 30° about, varies slightly. Good central fixation R. E.

October 23, 1900.—Retinoscopy under atropine shows refractive error R. E. + 4 D sph. + 1 D cyl. ax.

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used certain abbreviations, some of which may require explanation, *e.g.*, "C. S. R. E. 22°" means "constant unilateral convergent squint, the angle of deviation being 22° in distant vision, and the right being the deviating eye." "V. R. E." means "vision of the right eye." After glasses had been ordered, the angle of deviation and the visual acuity were always measured with the glasses on.

vert., L. E. + 4 D sph. Ordered glasses 0·5 D less than the retinoscopy. Ordered guttæ atropinæ 1 per cent. L. E. *only* every morning.

December 4, 1900.—Child uses unatropised R. E. always, both in near and distant vision, and squints with atropised L. E. Ordered, stop the drops and come again in a month.

January 8, 1901.—Child has now convergent squint. 18° with the glasses. The squint alternates. Ordered, wear glasses and come first week in April.

January 3, 1902.—Child has not been seen for a year. Soon after last visit mother says he lost his glasses. He has had no treatment since. There is now convergent squint R. E. 32°. R. E. has lost the power of central fixation. With L. E. bandaged, child sees the 1½-inch ivory ball when it is rolling on the floor, but cannot find it when stationary, unless it is close to his feet.

CASE A, 432. *January* 11, 1900.—Boy, aged 2 years 2 months, was brought to me at West Ham Hospital. He had alternating convergent squint 27°. Squinted more in near vision. Mother said he had squinted less than a month. I ordered ung. atropinæ thrice daily, for both eyes, and told the mother to bring him again in a week to have his eyes tested for glasses.

November 7, 1901 (one year and ten months later). —The mother did not use the ointment and come again as directed, as the child's father "did not believe in having the eyes messed about with." Child now has convergent squint L. E. 34°. L. E. has lost the power of central fixation, and the ivory-ball test shows V. L. E. to be considerably less than $\frac{6}{60}$.

CASE D, 730. *July* 13, 1901.—Girl, aged 7 weeks, has convergent squint R. E. 10° approximately, angle varies slightly. Good central fixation R. E.

July 17.—Retinoscopy under atropine, each eye + 2·5 D sph. Ordered, ung. atropinæ for L. E. *only*, every morning.

July 31.—Child uses R. E. (unatropised) almost as often as L. E. (atropised). Ordered, continue atropine L. E. only.

August 28.—Atropine has not been used. R. E. now converges 30° about, fairly constant in degree. R. E. has lost the power of central fixation. Ordered, continuous occlusion L. E. for three days.

August 31.—No fixation R. E.

I did not care to order continuous occlusion of this very young infant's better eye for many weeks, for fear of rendering it amblyopic. I therefore ordered the eye to be bandaged half of each day. In six weeks R. E. had regained steady central fixation. Probably continuous occlusion would have restored central fixation in one week.

Subsequent treatment was carried out, and eventually a perfect cure resulted.

CASE B, 24. *February* 4, 1896.—I was asked to see a girl aged 2 years 10 months. L. E. had squinted occasionally since she had whooping-cough, aged $2\frac{1}{2}$ years, but for the last four or five weeks the squint had been constant. Angle of convergence 26°. After instilling atropine into R. E. only for about an hour, she turned in R. E. and used L. E. always, both in near and distant vision. She could easily see a small marble at the other end of the room with L. E. The sight of this L. E. must have been perfect or nearly so.

A week later, retinoscopy under atropine.—R. E. + 4·5 D, L. E. + 5·5 D.

I ordered spectacles for constant wear, R. E. + 4 D sph., L. E. + 5 D sph. I also ordered a drop of atropine to be put in R. E. *only* every morning, and proposed exercises with the amblyoscope in a few weeks.

The parents, not being favourably impressed by my methods, determined to have "another opinion." The child was taken to an ophthalmic surgeon who said she was too young for glasses. He ordered atropine for *both* eyes twice daily. This was kept up for rather more than a year, after which she was given spectacles. At the age of seven years the surgeon operated on the left eye (tenotomy).

December 17, 1901.—The child was brought to me five years and ten months after I saw her first. She had convergence of L. E. 14°, while wearing her glasses. L. E. was prominent and caruncle sunk. The L. E. had lost the power of central fixation, and its vision was reduced to the counting of fingers at one foot from the face. I have since removed the deformity by advancement of the left external rectus, but the eye remains hopelessly blind.

Case B., 83. *October* 27, 1897.—A girl, aged 2 years, was brought to me. The mother said she had squinted occasionally for several months, but she had squinted constantly since a few days before the August bank holiday. She had convergent squint L. E. 21°. Using R. E. she could always see the half-inch ivory ball at the far end of the (22 feet) room. When R. E. was bandaged she could see the 1½-inch ball with the L. E., but she had great difficulty in finding the 1-inch ball unless she was allowed to begin to run after it before it had stopped rolling.

November 1, 1897.—Retinoscopy under atropine shows error of refraction to be, each eye + 3·5 D sph. + 0·75 D cyl. ax. vert. Ordered, spectacles 0·5 D less than the retinoscopy; continuous occlusion of R. E. for seven days; after that, one drop of liquor atropinæ to be put into R. E. only every morning.

December 7, 1897.—Child uses R. E. (atropised) in distant vision, and L. E. (unatropised) in near vision. Angle 17° with glasses. Ordered, continue.

February 8, 1898.—Child now uses (unatropised) L. E. always, both in near and distant vision, and squints always with (atropised) R. E. Ordered, stop using the atropine and come again in a month.

June 29, 1899 (one year and four months later).—Soon after the last visit the child's father, a bank manager, was transferred to a post in the north of England. The mother said that, as she was not able to bring the child to me, she thought she had better continue the drops. She used the drops, for the R. E. only, every day for about six months. Not since. The child has now constant squint R. E. 11°, with the glasses. Ivory-ball test shows vision of L. E. to be perfect, but vision of R. E. is barely $\frac{6}{60}$. Central fixation is present in R. E. but unsteady.

Of course all possible means have since been used to restore the sight of the R. E. The child has now learnt to read. On November 8, 1901, the vision of the L. E. (which at first squinted) was $\frac{6}{6}$, and the vision of the R. E. (which was at first the fixing eye) was $\frac{6}{12}$.

Case D, 332. On May 9, 1900, I saw, at Moorfields, a boy aged 3 years 2 months. He had squinted constantly, R. E., since the age of 2 years 8 months. No heredity. Abversion perfect. C. S. R. E. 46°. Good central fixation R. E. Easily sees half-inch ivory ball at about 20 feet with R. E. Ordered, atropine both eyes for retinoscopy.

May 16, 1900.—C. S. R. E., with atropine, 37°. Retinoscopy R. E. + 7 D sph. + 1·25 D cyl. ax. vert. L. E. + 6·25 D sph. + 1·5 D cyl. ax. vert. Ordered, glasses 0·5 D less than retinoscopy; also, guttæ atropinæ 1 per cent. L. E. *only* every morning. To return in one month.

June 5, 1901 (thirteen months later).—Mother used drops for L. E. for a month, but was then ill, so child has since been neglected. The glasses have been worn

constantly. C. S. R. E. 36°. R. E. has lost the power of central fixation. Ordered, continuous occlusion L. E. for one month.

July 3, 1901.—Central fixation, R. E., regained. Ordered, guttæ atropinæ 1 per cent. L. E. *only* every morning for two months.

August 28, 1901.—Patient uses L. E. (atropised) in distant vision, and R. E. (unatropised) in near vision. Ordered, continue drops L. E. *only*, for two months.

December 4, 1901.—Drops have been used until three weeks ago, not since, as they were all finished. Child now, without atropine, uses the originally squinting R. E. always, and squints constantly with the originally fixing L. E.

April 2, 1902.—Last time I accidentally omitted to give the mother written directions; there was, therefore, some misunderstanding. Child now squints constantly with L. E. (the originally fixing eye) 24°. L. E. has central fixation, but very unsteady. Ordered, guttæ atropinæ 1 per cent. R. E. only, every morning for one month.

May 7, 1902.—Child will not use (unatropised) L. E. voluntarily even in near vision. Ordered, continuous occlusion R. E. for one month.

June 4, 1902.—Steady central fixation L. E. Ordered, discontinue pad and use guttæ atropinæ R. E. only, for two months.

August 6, 1902.—Child now uses L. E. (unatropised) and squints with R. E. (atropised), always in near vision, and usually in distant vision also. Ordered, stop drops.

August 30, 1902.—Child now uses either eye indifferently, alternating convergent squint 24°, with glasses.

Case D, 286. *October* 9, 1900.—Girl, aged 5 years 7 months. She began to squint suddenly at the age of 3 years 9 months, and had squinted constantly

ever since. Already under atropine. C. S. L. E. 36°. Retinoscopy, R. E. + 4·5 D sph. + 0·25 D cyl. ax. horiz.; L. E. same at approximate macula. Fixation lost L. E. Ordered, glasses + 4 D sph. each eye. R. E. to be continuously occluded for one month.

August 9, 1901.—Case has been neglected, except that glasses have been worn. V. R. E. $\frac{6}{6}$, V. L. E. $\frac{4}{60}$. No fixation L. E. Ordered, continuous occlusion R. E. for three weeks.

August 30, 1901.—Treatment carried out. Good central fixation L. E. V. L. E. $\frac{6}{18}$. Ordered, guttæ atropinæ R. E. *only*, for six weeks.

October 4, 1901.—V. L. E. $\frac{6}{9}$. C. S. 16°. Child uses L. E. (unatropised) in near vision and R. E. (atropised) in distant vision. Ordered, continue atropine R. E. only, for two months.

December 3, 1901.—Vision $\frac{6}{6}$ each eye. Child uses L. E. and squints with R. E. always, both in near and distant vision. Ordered, stop atropine and come in two weeks.

May 6, 1902.—Child has not been seen for five months. She has squinted constantly with R. E. (the originally fixing eye) during that time. V. R. E. $\frac{6}{18}$, V. L. E. $\frac{6}{6}$. Amblyopia to $\frac{6}{18}$ acquired in R. E. since last December. Ordered, guttæ atropinæ L. E. only, for one month.

June 6, 1902.—Squints now with L. E. (atropised) 18°. V. $\frac{6}{6}$ each eye.

NOTE.—This case is altogether exceptional, on account of the age (nearly seven years) at which amblyopia in the R. E. was acquired.

Amblyopia ex anopsia, like congenital amblyopia, concerns almost entirely the central and paracentral region of the retina, and produces no contraction of the peripheral limits of the field of vision. But the blindness often reaches an extreme degree

which is never met with in the congenital form. In congenital amblyopia the central vision is never lower than $\frac{6}{60}$, the visual acuity normally found at 5° from the fixation point. In an extreme case of acquired blindness, on the other hand, there is often a scotoma extending about 15° to 20° round the centre of the field of vision. In this scotoma there may be bare perception of light. Outside this area fingers may be counted a foot or two from the face.

Amblyopia in Cases of Convergent Squint.

Congenital and acquired amblyopia having been studied separately, one is now in a position to discuss the cause of the blindness so often found in cases of unilateral convergent squint. In any individual case, seen for the first time when the squint has lasted several years, it is impossible to say how much of the blindness may be due to disuse of the deviating eye, and how much may be congenital. Statistics, however, enable one to draw a very accurate general conclusion.

Tables iii., iv., and v., show the visual acuity of the deviating eye in cases of constant unilateral convergent squint. I used Snellen's types or the ivory-ball test at the first visit, when possible, and on many subsequent occasions. I have since confirmed the results of the ivory-ball test by Snellen's types. The visual acuity noted in the tables is the final result, with optical correction, and after all

possible means had been used to remove any acquired amblyopia.

I have included in the tables only cases in which I could be reasonably certain as to the time of onset of the deviation, and in which my directions were subsequently followed to my satisfaction. The cases had either received no treatment before I first saw them, or they had merely been given glasses. Some had been operated upon.

Any defect of vision found in the cases in table iii. may be considered as congenital. In these recent cases any amblyopia which might have been acquired would certainly have been removed by the subsequent treatment.

Table iv. shows the visual acuity of the deviating eye after all possible means had been used to remove any defect which might be present. The vision was at first, in many cases, very considerably lower than shown in the tables.

In the cases in table v. the squint had, in most cases, lasted so long that no improvement in vision was possible.

A comparison of table iii. with table v. shows that congenital amblyopia only occurs in a very small proportion of the cases, and is never responsible for the extreme blindness so often found in neglected cases of unilateral squint.

TABLES SHOWING THE FINAL VISUAL ACUITY, AFTER ALL POSSIBLE MEANS HAD BEEN USED TO IMPROVE IT, OF THE DEVIATING EYE IN 985 CASES OF CONSTANT UNILATERAL CONVERGENT SQUINT.

TABLE III.

Cases which I saw first when the patient had squinted constantly during less than one-eighth of his or her life.

Vision of the Deviating Eye.	Age of Onset of the Deviation.			Total.
	Before 12 Months.	1 to 3 Years.	After 3 Years.	
$\frac{6}{6}$	23	62	80	165
$\frac{6}{9}$ and $\frac{6}{12}$ ...	2	6	9	17
$\frac{6}{18}$ and $\frac{6}{24}$ ...	1	3	5	9
$\frac{6}{36}$ and $\frac{6}{60}$ ...	0	1	1	2
Less than $\frac{6}{60}$ ...	0	0	0	0
Fixation lost irrecoverably ...	0	0	0	0

TABLE IV.

Cases which I saw first when the patient had squinted constantly during more than one-eighth and less than one-half of his or her life.

Vision of the Deviating Eye.	Age of Onset of the Deviation.			Total.
	Before 12 Months.	1 to 3 Years.	After 3 Years.	
$\frac{6}{6}$	5	17	51	73
$\frac{6}{9}$ and $\frac{6}{12}$ ...	3	26	32	61
$\frac{6}{18}$ and $\frac{6}{24}$ ...	0	14	14	28
$\frac{6}{36}$ and $\frac{6}{60}$ ...	0	5	9	14
Less than $\frac{6}{60}$ ...	0	1	4	5
Fixation lost irrecoverably ...	0	2	5	7

Table V.

Cases which I saw first when the patient had squinted constantly during more than one-half of his or her life.

Vision of the Deviating Eye.	Age of Onset of the Deviation.			Total.
	Before 12 Months.	1 to 3 Years.	After 3 Years.	
$\frac{6}{6}$	0	3	11	14
$\frac{6}{9}$ and $\frac{6}{12}$ ...	2	7	19	28
$\frac{6}{18}$ and $\frac{6}{24}$ ...	4	32	54	90
$\frac{6}{36}$ and $\frac{6}{60}$ ...	8	53	41	102
Less than $\frac{6}{60}$ ...	55	103	21	179
Fixation lost irrecoverably ...	56	110	25	191

When I have been consulted about a case of squint, I have always warned the parents of the patient that, in the event of a younger member of the family developing a squint, the case ought to receive attention without delay. I have, therefore, been fortunate enough to see an unusually large proportion of my cases soon after the first appearance of the deviation.

It is remarkable that only eight cases, commencing before twelve months of age, appear in table iv. This is evidently because parents who have been warned, and those who are especially solicitous for the welfare of their children, seek advice immediately. These cases are included in table iii. The other infants are usually left without treatment for several months, so that they appear in table v.

CHAPTER VI

THE METHOD OF INVESTIGATING A CASE OF SQUINT

EVERY case of squint should be systematically investigated, as it is only by a thorough knowledge of each case that a rational line of treatment can be determined upon. The following is the plan I always use. It may appear rather formidable at first sight, but, with practice, one can carry out the various tests with great rapidity and precision. The time will surely not be grudged when it is remembered that, in cases which are presented early enough, the patient's whole future career may depend upon the skill and care of the surgeon who first sees the case.

(1) History.

(2) The character of the squint.

(3) The power of fixation in the deviating eye.

(4) Movements of each eye separately. Dynamic convergence.

(5) Vision testing.

(6) The condition of the fusion faculty.

(7) The angle of the deviation.

After using atropine for from three to eight days :—

(8) The refraction.

(1) HISTORY.—Under this head should be noted: (*a*) Age of onset, when it can be determined. It may often be fixed very precisely by reference to some family event, such as the birth of the next child. Now that general practitioners and the laity are becoming more alive to the importance of early treatment, one sees a larger proportion of the cases soon after the first appearance of the deviation than formerly. (*b*) Mode of onset. Whether it began as an occasional squint or was constant from the first. (*c*) Any illness or injury immediately preceding the appearance of the deviation, *e.g.*, whooping-cough, measles, a blow on the head, "fits," severe fright, etc. (*d*) Evidence of heredity—squint in brother, sister, parent, grandparent, or in more than one other near relation.

(2) THE CHARACTER OF THE SQUINT.—The presence of a deviation and its character, whether convergent or divergent, unilateral or alternating, may often be determined by simple inspection. But appearances are sometimes misleading, *e.g.*, the high angle gamma often found in hypermetropes may simulate a divergent squint or mask a slight convergent squint. The low, or even negative, angle gamma usually associated with myopia may give an appearance of abnormal convergence or mask a slight divergence.

The cover test is at best only a rough test having many sources of error. It is, moreover, not possible

to use it with young children. But, as it is very generally employed, it will be well to describe the proper method of making the test, and the fallacies to be guarded against.

Tell the patient to look steadily at some distant object. Take a narrow card or folded paper and, with a rapid lateral movement, cover, say, the patient's left eye, taking care not to touch his face. If the right eye makes no movement it was probably[1] not squinting. Now uncover the left eye, and see that the patient looks steadily at the distant object. Screen the patient's right eye in the same way. If the left eye makes no movement it was probably[1] not squinting either. If, however, when one eye is covered, the other has to make an outward movement in order to fix the object, it was previously squinting inwards; if it rotates inwards it was squinting outwards.

In a case in which the patient has been shown to have a squint, but in which he can fix with either eye at pleasure without the other eye being screened, and can maintain fixation after a momentary closure of the lids, the squint is alternating.

In some occasional squints and heterophorias, while both eyes fix truly when uncovered, either eye deviates when screened, but immediately recovers itself on removal of the screen.

The mirror test.—This is an entirely satisfactory test, and can be used quite well even in the case of the youngest infants. The patient should be in the dark room, with the lamp behind him. The light is reflected from the mirror of an ophthalmoscope, from a distance of about two feet, into

[1] If the eye has lost the power of central fixation, it may perhaps make no movement, or it may move in a direction which would mislead any but a careful observer.

the patient's eyes. An infant will immediately look at the mirror; an older patient should be told to do so. A tiny image of the ophthalmoscopic mirror is formed on the patient's cornea. Owing to the angle gamma, this reflection of the mirror is usually slightly to the nasal side of the centre of the pupil. By flashing the light rapidly from one eye to the other, any want of symmetry in the position of the reflections is at once detected. It may easily be seen, too, which is the deviating eye, and, with practice, a very good guess as to the extent of the deviation may be made.[1]

Squint or paralysis.—In the case of a patient who is old enough to speak, the persistent diplopia would prevent one mistaking a case of paralysis or paresis of one or more of the external ocular muscles for a case of comitant squint.

But, in the case of an infant who has recently suffered from diphtheria, an objective test is required. The patient is on the nurse's knee in the dark room with the light behind him. The nurse holds his head immovable. The light is thrown into his eyes, from an ophthalmoscope mirror, from a position slightly to one side of him. When he fixes the mirror the approximate degree of his

[1] According to Hirschberg, when the reflection on the squinting eye is at the margin of the cornea the angle of the deviation is about 45°, when it is at the margin of an average-sized pupil the angle is about 15°. Allowance should be made for the estimated size of the angle gamma.

deviation is noted. The light is next thrown into his eyes from the other side. If, when he again fixes the mirror, the angle of the deviation is greater or less, we have to deal with a paralysis or paresis, and not with a comitant squint.

In carrying out the test, it is essential that the light be thrown into the eyes in both cases from approximately the same level, as, in many cases of true squint, the eyes converge more on looking down and diverge more on looking up.

(3) THE POWER OF CENTRAL FIXATION.—An exceedingly important point, in its bearing on the treatment and prognosis of a case of unilateral squint, is the presence or absence of the power of central fixation in the deviating eye.

The patient being in the dark room, with the light behind him, throw the light from an ophthalmoscope mirror first into his good eye, while he looks at the mirror. Note the position of the reflection of the mirror on the cornea of this eye. Then cover the good eye, and note whether the previously deviating eye can now fix the mirror so as to bring the corneal reflection into a corresponding position. If it does so, there is central fixation. There may be no fixation, in which case the eye wanders. Or there may be false fixation, in which case an eccentric part of the retina is used for the purpose. An eye which at first appears to have lost fixation may sometimes, with a little patience, be induced to fix correctly.

In the clinics one frequently sees some such procedure as the following: The surgeon covers the patient's good eye, then asks him to follow the movement of his finger with the deviating eye. If he is able to do so, it is assumed that he has central fixation in this eye. This is entirely fallacious. If the deviating eye has any sight at all it can usually follow the movements of a large object. If the deviating eye has false fixation in a position of only slight convergence, even the most prolonged examination by this method will certainly lead to error.

(4) THE MOVEMENTS OF EACH EYE SEPARATELY should be tested by covering one eye and getting the patient to look with the uncovered eye from side to side and up and down. The test is then repeated with the other eye. This may usually be done even in the case of young infants by showing them something in which they are likely to take an interest. If each eye can be separately abverted until the edge of the cornea touches the outer canthus, abversion may be considered full. The power of adversion varies considerably within normal limits. Most people can advert each eye separately until the corneal margin is within less than one-tenth of an inch of the caruncle.

DYNAMIC CONVERGENCE.—A careful distinction should be made between static and dynamic convergence, just as static and dynamic refraction are distinguished from each other.

When a person looks at a distant object, if he has no squint the static convergence of the eyes is *nil*. If he has a convergent squint there is a

static convergence corresponding to the angle of the squint. If he has a divergent squint, the static convergence is a negative quantity.

If now he fixes a near object, there is superadded to the static convergence a dynamic convergence.

A person who in distant vision has no squint will usually exercise just sufficient dynamic convergence in near vision (in association with dynamic refraction, or accommodation) to allow him to fix the object correctly with both eyes. If, however, his dynamic convergence be excessive, the eyes will tend to squint inwards in near vision. A perfect fusion sense will keep this tendency in check. But if the fusion sense be defective, this tendency will be free to cause a squint in near vision. In a case of divergent squint, when the fixing eye accommodates for a near object the divergent eye will usually recover itself to a certain extent. In convergent squint, the faulty eye should turn in still more towards the nose in near vision.

Method of estimating the dynamic convergence in a case of squint.—Stand at arm's length from the patient. Shut one eye and hold the point of the finger, or perhaps some more attractive object, in line with the open eye and the patient's fixing eye. Let the patient look at the object while it is gradually brought nearer his fixing eye. The fixing eye will remain immobile, the deviating eye manifesting the convergence of both. In this way, the slightest movement of convergence of the deviating

eye can at once be seen, and the point at which the deviating eye begins to diverge again can be accurately noted. This simple procedure should never be omitted.

(5) VISION TESTING. — (*a*) The vision of patients who are old enough to read letters should be tested with *Snellen's distant types*. This would appear a very simple matter, yet it is surprising how often, in the clinics, one finds the vision of the better eye recorded as the vision of each eye, even by experienced assistants. The desire to use the accustomed eye is so strong that, when this is shaded, the patient will involuntarily screw his head round to try to see past the shade.

(*b*) *Ivory-ball test for young children.*—Apart from its scientific interest, it is often of great practical importance to be able to estimate approximately a young child's visual acuity. For this purpose I use five little ivory balls varying in size from half an inch to one and a half inches in diameter. The child is first allowed to handle the balls with both eyes open. Then one eye is covered by a pad, or, if he wears glasses, by a piece of cotton wool stuffed behind the lens. He is then asked to go and pick up the balls as they are thrown on the floor to a distance of six or seven yards, one by one, beginning with the largest. By spinning the ball in the fingers as it is thrown, it can be made to "break" on touching the floor, so that it does not go quite in the direction in which

it appeared to have been thrown. It is easy to tell, by the way in which the child runs for the ball, whether he really sees it before he starts or is only going to look for it. I test the presumably better eye first, so as to give the other eye the benefit of experience.

Children are always ready to play this ball game. This method of vision-testing only takes a few minutes, and it succeeds with most children who are old enough to walk. I have used it since 1896, and, in cases in which I have subsequently been able to test the vision by means of Snellen's types, I have found my conclusions confirmed.

The test has also another use. In a case of constant unilateral squint which has been neglected or inefficiently treated, the deviating eye may have become highly amblyopic. The parents know nothing of this, owing to the fixing eye having good sight. The treatment to be undertaken with a view to curing this blindness will need great care and perseverance on the part of the mother and nurse, and may be long and tedious. Nearly all mothers will carry out the prescribed treatment intelligently and devotedly, but a few seem extraordinarily callous about anything which does not directly concern themselves and which does not lie within their limited range of comprehension. The matter is too important to be left in doubt, for the child's whole future career may depend upon it: and in any case ocular demonstration of results is

encouraging. In a case of amblyopia in a young child I demonstrate to the mother, with the ivory-ball test, the vision at each visit.

(6) THE CONDITION OF THE FUSION FACULTY may, as a matter of convenience, be investigated at this point, but it is usually better to defer the examination until the state of the refraction has been ascertained and the effect of the mydriatic has passed off.

Examination with the *amblyoscope* I have found by far the most rapid, precise, and reliable method of ascertaining the condition of the fusion sense. It is fully described in chapter viii.

I wish again to draw attention to the distinction between the possession of the fusion faculty and the presence of binocular vision. A patient may have a convergent squint of very high degree, with suppression of the vision of the deviating eye, and yet training with the amblyoscope may have strongly developed his fusion faculty. In this case, if the patient looks into the amblyoscope while the instrument is adapted to the angle of the squint, he fuses the pictures—his fusion faculty finds its expression in the act of binocular vision. The same thing happens when his eyes are put "straight" by operative or other means.

Some information may be gained as to the "depth of suppression" from the ease or difficulty with which diplopia may be artificially elicited. Diplopia may sometimes be elicited by placing a red glass before

the better eye and alternately covering and uncovering it while the patient looks at a candle flame. There is a fallacy here which must be guarded against—the patient recognises the fact that he sees a red light with the "good" eye, and that, when this is covered, he sees a white light. He therefore often says he sees two lights, a red and a white, even though he does not see them both simultaneously. If the red glass fails to produce diplopia, success may often be attained by placing a *horizontal* prism before the deviating eye, so as to throw the image of the flame on a part of the retina nearer the macula.

Some writers have recommended, as a test, placing a prism *vertically* before one eye, to throw the false image out of what they call the "band of suppression." Diplopia can almost always be elicited in this way, even in cases of total absence of the fusion sense. The explanation of this is not far to seek. The conjugation of the two eyes in horizontal movements, being intended to subserve the act of binocular vision, was probably acquired at no very early period in the development of the human race. The conjugation of the eyes in vertical movements, on the other hand, I believe we share to a great extent with most of the mammals. It is not surprising that disturbance of this very ancient function, by vertical displacement of the image in one eye, should produce diplopia.

But diplopia tests really give very little assistance in foretelling the result of fusion training. Some cases, in which diplopia is with difficulty elicited by these methods, eventually get the highest grade of binocular vision. Others, again, who easily see double, may be incapable of acquiring more than grade I binocular vision.

(7) THE MEASUREMENT OF THE ANGLE OF THE DEVIATION.—At the first, and at each subsequent, visit the angle of the deviation is accu-

rately measured. When the child has been ordered glasses, the measurement is always made with the glasses on. The case-sheet then shows at a glance the progressive effect of the treatment upon the deviation, and helps one to decide whether it is advisable to supplement this treatment by operation.

Four methods of measuring the angle of deviation are here described.

(*a*) *The deviometer.*[1]—This instrument can be very rapidly used, no adjustment being required. Measurements obtained by it are very accurate. It can be used quite easily even with the youngest children.

A wooden stand, about 10 inches high, supports a horizontal wooden arm, 2 inches wide, $\frac{1}{4}$ inch thick, and about 2 feet long. This arm is pivoted at the end, so that it may be swung over to either side as required. The arm is painted black in front. On the back is a scale of tangents to degrees at 60 centimetres distance. A flat, hook-shaped piece of brass, having a white spot on it, slides along the arm. In front of the pillar, below the zero of the scale, is a specially made electric lamp, 5 inches high and $\frac{3}{4}$ inch in diameter. Flexible wires go from the electric lamp to the

[1] I have no wish to claim originality for this instrument. The principle is that of Maddox's scale and Priestley Smith's tape, the former of which I used for years before I devised the deviometer.

wall plug. An electric bell-push is used instead of a switch, so that, by pressing the button, the light may be flashed on and off very rapidly. A string 60 centimetres long is attached to the upright pillar of the instrument. At the end of the string is a ring.

Measurements and details of construction of the deviometer will be found in the Appendix.

The instrument is put on a table. The nurse sits at the table with the child on her knees. She puts the ring on her finger, and holds the child's head steady with her hands, keeping the string taut. The surgeon, keeping his observing eye close above the top of the stand, "sights" the child's eyes over the nick in the top of the stand, and presses the button. The child immediately looks at the light with his fixing eye. The reflection of the lamp forms a vertical line of light on the cornea of this eye, which shows the correct position of fixation. The position of the line of light on the cornea of the squinting eye enables a good guess to be made as to the angle of the deviation. The light is discontinued. The brass traveller with the white disc is slid along the arm to the position which corresponds to the guess. The brass traveller being tapped with the finger, the metallic sound causes the child to look at it. If it does not, a lighted match, held in front of the traveller, will always attract his attention. The button is then pressed, and the light flashed on

for an instant. If the line of light on the cornea of the squinting eye is in a corresponding position to that which it formerly occupied in the fixing eye, the angle of the squint is read off on the scale on the back of the arm. If not, the traveller is moved a little, and when the child looks at it the

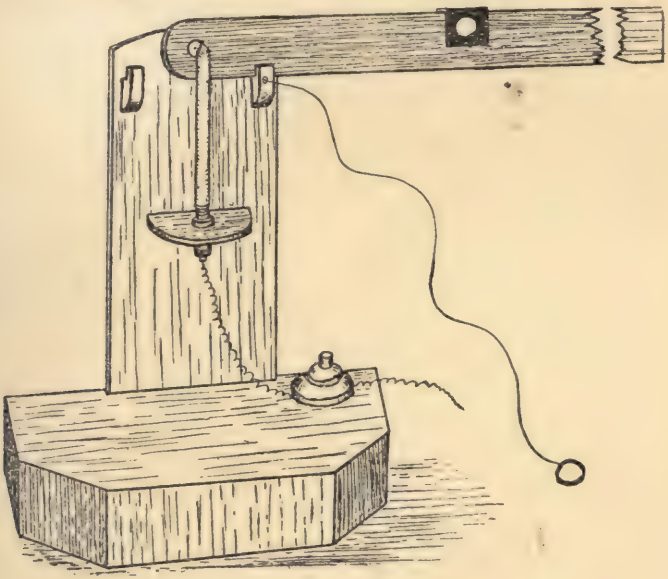

FIG. 9.

light is flashed on again, and so on till the true position is found.

An older patient can, of course, sit at the table and hold the string himself, and look at the zero of the scale, or the white disc on the traveller, when directed to do so.

(*b*) *Maddox's tangent scale* is an admirable device for measuring the deviation. The method is very rapid and accurate. It is not, however, easy to use with small children. I use it at Moorfields, because the apparatus is fixed in the wall out of harm's way, and there is nothing to get out of order, however roughly it may be used.

The large figures represent tangents to degrees at 5 metres. They are useful for measuring the strength of prisms. With these we are not at present concerned. The small figures, in the horizontal scale, represent tangents to degrees at a distance of one metre. They are printed on a strip of paper, which is pasted on a board about seven feet long. In the centre of this scale is a candle. Below the candle is attached a light bamboo rod one metre long.

The patient rests his cheek against the end of the metre rod. The surgeon puts his head below the rod, so that his eye is vertically below the rays of light which pass from the candle to the patient's face. The patient is first told to look at the light. The position of the image of the candle flame on the cornea of the fixing eye is noted. A guess is made as to the angle of the squint. The patient now is told to look at the figure which represents the guess. If this is too much or too little, other figures are named till the reflection of the candle flame on the cornea of the deviating eye occupies

a position similar to that which it formerly occupied in the fixing eye.

(*c*) *Priestley Smith's tape method.*—The advantages of this method are that it takes very little time and the apparatus required is simple. It is moderately accurate. It is, however, not easy to use with young children.

A string one metre long has a ring at one end. To the ring is attached a graduated tape. The tape has a weight at its other end. The patient holds the free end of the string against his temple. The surgeon puts the ring on a finger of one of his hands in which he holds an ophthalmoscope mirror. The tape is allowed to slide between the fingers of the other hand, the weight keeping the tape taut. The patient is first told to fix the mirror, while the light of a lamp is reflected into the fixing eye. The position of the image of the mirror, on the cornea of the fixing eye, is noted. The light from the mirror is now thrown on to the deviating eye, and the patient is directed to look at the surgeon's tape hand. This is moved horizontally, till the position of the image of the mirror, on the cornea of the squinting eye, is similar to that which it formerly occupied on the cornea of the fixing eye. The string keeps the ophthalmoscope hand at one metre from the patient's eye. The observer keeps the tape hand as nearly as possible at the same distance from the patient's eye. The graduated scale on the tape, where it slides through the

tape hand, shows approximately the angle of the deviation in degrees.

If the length of the string be made 60 cm. instead of one metre, an ordinary Continental tape measure may be used for the graduated tape, one centimetre representing approximately one degree.

(*d*) *The perimeter method.*—I describe this because a perimeter is to be found in every eye clinic and ophthalmic surgeon's consulting-room. The patient is seated at the perimeter, which is adjusted so as to bring his deviating eye *accurately* in centre of the arc. A candle is placed at the far end of the room, in line with the zero of the perimeter and the patient's deviating eye. He is told to look steadily at this candle with his fixing eye. A second candle or taper, with the eye of the surgeon looking exactly over the top of the flame, is carried round the arc of the perimeter till the reflection of the flame lies in the centre of the cornea of the deviating eye. The position of the taper on the graduated arc of the perimeter shows the angle of the squint in degrees.

This is an unsatisfactory method. It is inaccurate, as it takes no account of the angle gamma. It cannot be used for young children. The preliminary arrangement takes up so much time that a surgeon who relies upon this method is apt to neglect to measure the squint at all.

The angle gamma may be measured separately, if the patient's squinting eye has not lost the power of

central fixation. Cover the fixing eye. Let the patient steadily fix the zero of the perimeter with the squinting eye. The taper, with the surgeon's eye looking exactly over it, is carried along the arc till the reflection of the flame appears in the centre of the cornea. The position of the taper on the graduated arc shows the size of the angle gamma. This angle gamma should be added to the perimeter measurement of a convergent squint and subtracted from that of a divergent squint.

(8) THE REFRACTION.—This is examined by retinoscopy, after atropine has been used, for both eyes, three times a day, for from three to eight days. For young children I prefer the atropine ointment, 1 per cent. Atropine drops, if used too freely, occasionally cause unpleasant symptoms, whereas the ointment almost never causes trouble. The nurse or mother should be shown how to insert the ointment within the lower eyelid with a glass rod. Frequently in an infant atropine produces perfect cycloplegia with only very slight mydriasis.

Children of two years and upwards may, with a little tact, usually be induced to allow one to put on a trial frame and proceed with retinoscopy in the usual way. If there be astigmatism, one should put in the frame the lens which turns the shadow in the lowest meridian and the cylinder which represents the degree of astigmatism. The correct position of the axis of the cylinder is then found by manipulating it until the shadow is even

all round. I habitually use this method in examining the refraction of the eyes of adults without a mydriatic.

In the case of an infant, the nurse sits in the dark room with the child in her arms. The light is placed above and behind the child's head. The light being reflected from the ophthalmoscope mirror into the child's fixing eye, he immediately looks at the mirror. Holding lenses at arm's length before the child's fixing eye, one can measure the refractive error in the highest and lowest meridians. One can estimate fairly accurately the axis of the cylinder, though some slight readjustment may subsequently be required when one is able to repeat the retinoscopy with a trial frame on the child's face. If the deviating eye still retain its power of central fixation, one may induce it to fix during the performance of retinoscopy upon it, by obscuring the view of the fixing eye with the hand which holds the lenses. If fixation have been lost, an approximate correction should be ordered, to be replaced by an accurate correction, if and when one succeeds in restoring this function.

In the case of an older patient, one can measure very accurately the refractive error of an eye which has lost fixation. Throw the light into the fixing eye, and note the position of the reflection of the mirror on the cornea, while the patient fixes the mirror. Now direct the patient to look at a small

object, which is held three or four feet away from him by an assistant. Throw the light on to the cornea of the deviating eye, and manœuvre the object till the reflection of the image on the cornea is in a position corresponding to that which it formerly occupied in the fixing eye. Now proceed with the retinoscopy.

CHAPTER VII

THE TREATMENT OF CONVERGENT SQUINT

I PROPOSE to describe first the treatment I adopt in cases of constant unilateral squint. Any modification of treatment required in occasional or alternating cases will be considered afterwards.

The objects to be kept constantly in view in the treatment of squint are—(*a*) To prevent deterioration of the vision of the deviating eye, and to restore, as far as possible, the sight of this eye in cases in which amblyopia from disuse has already been allowed to occur. (*b*) To endeavour to remove the fundamental cause of the squint, by training the fusion sense at the earliest possible age. (*c*) To restore the visual axes to their normal relative directions.

There are five therapeutic measures at our disposal, any or all of which it may be necessary to use in our endeavour to attain these objects. (1) Optical correction of any refractive error which may be present. (2) Occlusion of the fixing eye. (3) Instillation of atropine into the *fixing* eye *only*. (4) Training the fusion sense. (5) Operation.

(1) Optical Correction.—As has been demonstrated in chapter iv., the essential factor which allows a deviation to occur is a defect of the fusion faculty. The eyes then, being uncontrolled by the necessity for fusion, are for a time kept approximately "straight" by their motor co-ordinations. But they are in a state of unstable equilibrium, and are ready to squint, either inwards or outwards, in response to influences which would have no effect if the fusion faculty were normal. In a very large proportion of the cases it is the state of the refraction which chiefly determines whether the eyes shall deviate inwards or outwards. Thus, in the great majority of cases, the eyes of hypermetropic squinters deviate inwards, and the eyes of myopic squinters deviate outwards. It is rational treatment, therefore, to attempt to overcome the deviation by optical correction of any refractive error which may be present.

In cases of simple hypermetropia, or hypermetropic astigmatism, or compound hypermetropic astigmatism, my usual practice is to order spectacles fully correcting any astigmatism which may be present, and correcting all but 0·5 D of the hypermetropia. The reason for the slight under-correction of the hypermetropia is this: When the effect of the atropine, used for the retinoscopy, has passed off, some of the hypermetropia will in any case become "latent," so that fully correct-

ing glasses, which gave perfect distant vision under atropine, will, when its effect has passed off, make all distant objects appear misty. This blurring of distant objects not only shortens the child's range of vision at a time when the acuity of the physical senses has the most marked effect upon his mental development, but the effort to see distinctly actually appears to affect unfavourably the angle of the deviation.[1]

In a case of mixed astigmatism, the refractive error should be exactly corrected.

A certain percentage of cases of convergent squint are myopic. Any myopic astigmatism should of course be exactly corrected. But as regards simple myopia, it might naturally be thought that a considerable under-correction would tend to lessen the abnormal convergence by preventing any effort of accommodation even in near vision. I began by acting upon this assumption, but was gradually forced by experience to abandon it. I find that the best results are obtained by exactly correcting any myopia and myopic astigmatism which may be present. Myopes who begin early to wear fully correcting glasses, use them

[1] I once took over a considerable number of hospital squint cases which had previously been under the care of a colleague, who used habitually to over-correct the hypermetropia to the extent of 1 D. In the majority of these cases the deviation became less within a few weeks of ordering glasses with which the patients could see distinctly.

quite comfortably for all purposes, and appear to have as good a range of accommodation as emmetropes.

In a case of anisometropia, the refractive error of each eye should be corrected according to the preceding rules. This applies even to cases in which one eye is hypermetropic and the other myopic.

When glasses are ordered, it is a good plan, especially with young children, to continue the atropine, which has been used for the retinoscopy, until the glasses arrive from the optician. It should then be discontinued. Even an infant soon discovers that he sees better with the glasses than without them, and by the time the effect of the atropine has passed off, the wearing of glasses has become as much a habit as the wearing of clothes.

The glasses should be worn constantly, except when the child is in bed at night. They should never be removed at any other time, except for toilet purposes.

In the case of children who are old enough to attend school, some surgeons order one pair of glasses for distant vision and a stronger pair for reading. I have tried this plan and found it most unsatisfactory. In changing from one pair to the other there is often a considerable interval in which no glasses are worn at all. Besides, the exercise of a *normal* amount of accommodation in association

with dynamic convergence, in near vision, is a physiological act, and its suspension has not appeared to me to have permanently lessened the angle of the squint.

The quality and fit of the spectacle-frames are very important. Steel is the best material for children. Steel frames of good quality will remain without rust for a long time, if the child be kept clean. After a time, the growth of the child's face will necessitate larger frames. For infants and young children it is a good plan to have the lenses oval with their long axes vertical, in order that there may be no temptation for the child to look over them. In a case of astigmatism it is generally better not to order circular lenses, lest they become rotated in the frames, displacing the axes of the cylinders. The lenses should be large, and should be centred for distant vision. They should be as close to the eyes as they can be without touching the lashes, and should be thick in order that they may not easily be broken. The bridge piece, which arches over the bridge of the nose, should be of tempered steel, broad, flat, and strong, and should be very accurately fitted. A broad thin plate of tortoise-shell, carefully fitted under the arch of the bridge piece, prevents rust and distributes the pressure over a larger area. Spectacles for children of three years and upwards should have flexible curl sides to hook behind the ears.

Infants and very young children should have their glasses tied on. The sides, in this case, should be straight and should have a loop at the end. They should be very short, only reaching to just above the ear. About $\frac{3}{4}$ inch near the loop should be wrapped with wool. The glasses are tied on with tapes, passed through the loops, behind the child's head. These frames are very comfort-

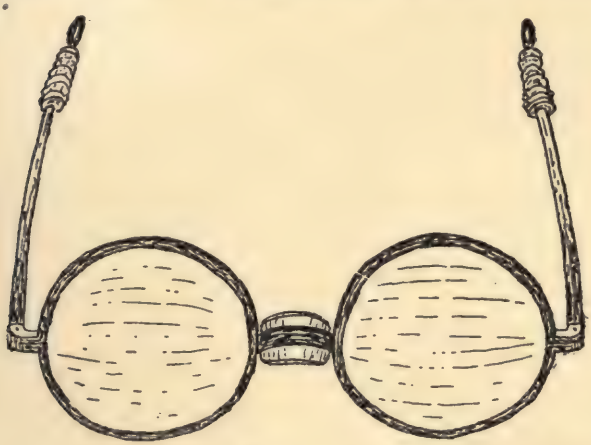

FIG. 10.

able. If the sides were of the usual length the tapes would act on the end of a long lever, causing pressure above the ear, and perhaps ulceration of the tender skin of the infant. When the infant is put down to sleep in the daytime these frames are not removed. These very short sides do not press on the pillow and lift the glasses from the child's face.

It is usually stated that children under three or

four years of age are too young for glasses.[1] No infant is too young to wear glasses should they be required. Many of my squinting patients have worn spectacles such as I have described, before twelve months of age, and some before six months.

Of course young children sometimes break their glasses, but I have never known a case in which the eye has been injured thereby.[2] The lens, being held in the frame, does not break into splinters but cracks across, or chips at the edge.

(2) Occlusion of the Fixing Eye.—In the case of a child who has squinted constantly with one eye for a considerable proportion of his life, and who has either received no treatment at all or who has been merely given a pair of glasses, one usually finds that the deviating eye has become more or less blind. If the ivory-ball test shows the visual acuity of this eye to be not less than $\frac{6}{36}$, the case may be treated at once in the manner described in paragraph 3. Often, however, one finds that the blindness has progressed far beyond

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[1] Since this was written a vast improvement in this respect has taken place, but the advance has not yet become universal.

[2] I have known an eye destroyed by a bullet from a small American pocket pistol, and another by a stone from a catapult. Glass was carried into the eye by the missile in each case. In the former, the thick spectacle lens probably saved the child's life; in the latter, it certainly did not contribute to the loss of the eye.

this point, so that the power of central fixation may have been lost and the central visual acuity reduced to the ability to count fingers close to the face, or, in some cases, even to bare perception of light.

In a young child, an attempt should always be made to restore, as far as possible, the sight of the deviating eye, by forcing the child to try to use it. For this purpose I order the fixing eye to be *continuously* occluded for a time. It is not a good plan to order the eye to be occluded for part of each day only; apart from the fact that this is not nearly so rapid and effective as continuous occlusion, the child usually cries every time the shade is applied, so that the treatment is seldom properly carried out. When the better eye is continuously covered, the child soon becomes accustomed to the shade, so that, after a day or two, he usually ceases to object to it.

Continuous occlusion of the better eye can only be carried out efficiently by the exercise of great care in applying the pad and constant supervision to prevent the child pulling it aside. For infants, a gauze pad secured by a few turns of bandage answers best. In the case of children who are beginning to get about, the gauze pad should be held in place with a ring of strapping-plaster. Children who are well cared for may have the pad changed every second or third day. It may, if desired, be left undisturbed for a week at a time without any harm resulting.

If the child is especially tractable, and has a reliable nurse in constant attendance, it may be sufficient, after the first day or two, to pack a gauze pad behind the spectacle lens. The child will require constant watching, or he will push the pad upwards and outwards and peep down the side of

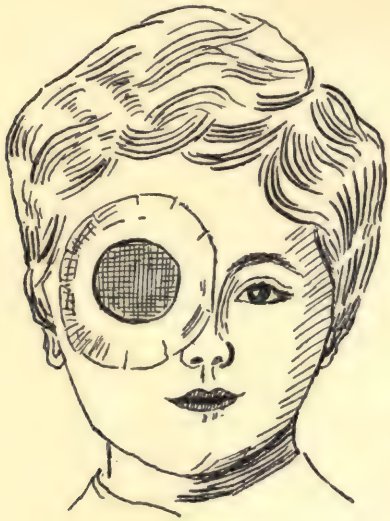

Fig. 11.

his nose. The gauze pad very firmly secured with strapping-plaster is the best plan in nearly all cases, and is the only possible method for hospital patients, whose parents are seldom able to give them undivided attention.

I examine the child again at the end of two or three weeks. If the vision of the deviating eye has improved sufficiently, the shade is discontinued

and the case treated as described in paragraph 3. If not, the fixing eye is occluded for another three or four weeks, after which the child is again examined. If occlusion of the fixing eye is going to do much good, one usually finds a very great improvement in the vision of the squinting eye within three weeks. If improvement has not taken place by the end of two months, the case is almost hopeless, though further persistence is sometimes rewarded with success.

(3) INSTILLATION OF ATROPINE INTO THE FIXING EYE ONLY.—Atropine has the property of temporarily paralysing the ciliary muscle, and so suspending the power of accommodation of the eye. When, therefore, atropine is instilled into a normal emmetropic eye, or an eye whose refractive error is corrected by glasses, this eye still sees distant objects clearly, but is unable to focus near objects. An unatropised eye whose vision is only one-sixth or even one-tenth of the normal is able to see objects, at the reading distance, more clearly than a normal eye whose accommodation is paralysed by atropine.

In the case of a young patient, the visual acuity of whose squinting eye has been shown by the ivory-ball test to be not much less than $\frac{6}{36}$, I order atropine to be put into the *fixing* eye *only* every morning. The child, of course, wears his glasses at the same time. He will be unable to see distinctly his toys and picture-books with the atropised

fixing eye. But he quickly discovers that, by making a conjugate lateral movement of both eyes until he has brought the squinting eye to bear on these near objects, he can see them much more clearly. He acquires the habit of always using the (atropised) better eye for distant vision and his (unatropised) worse eye for near vision. In this way the worse eye is most efficiently exercised, and amblyopia ex anopsia prevented. And, in a case in which a considerable degree of blindness has already been acquired, one finds at each visit, if the child be young enough, a steady improvement in vision. In many cases, after a few weeks or months of this treatment, the vision of this previously amblyopic eye becomes perfect, or nearly so. When the visual acuity of the (unatropised) "deviating eye" approaches the normal the child uses this eye always both in near and distant vision, and turns in the (atropised) "fixing eye" instead. I then stop the atropine for two or three weeks to see what will happen. Usually the child returns to his old habit of squinting with the eye with which he squinted at first, and fixing with the "fixing eye." In this case I order atropine every morning for the *fixing* eye *only* for the first seven days in each month. It occasionally happens, in the case of a young child, that, on discontinuing the atropine, what was originally the fixing eye continues to exhibit the deviation. The case may be left for three or four weeks, but care must be taken lest

this originally fixing eye now become amblyopic (see Case B, 83, and Case D, 332, chapter v.). The balance may easily be kept by, if necessary, atropising the other eye for a few days.

The use of atropine for the *fixing* eye *only* is an exceedingly efficient curative measure. A young child spends perhaps one-third of his waking hours in looking at near objects. So that when he uses the (atropised) "fixing eye" in distant vision and the (unatropised) "squinting eye" in near vision, this is equivalent to perfect occlusion of the "fixing eye" for several hours each day. And when his glasses are taken off for any purpose, if he has much refractive error, he turns in the atropised "fixing eye" and uses the unatropised worse eye, even in distant vision.

As regards the form in which the atropine is used, I usually order one drop of a 1 per cent. solution of atropine sulphate to be put into the fixing eye only, every morning. It may be inserted with a "dropper," or with a small camel-hair brush. Atropine ointment answers the same purpose. The lower lid should be drawn down, and a small piece inserted into the conjunctival sac with a glass rod. Lamellæ containing $\frac{1}{200}$ to $\frac{1}{100}$ of a grain of atropine sulphate may be used instead, but nurses usually find them rather difficult to insert.

I always see the child again within a month of beginning this treatment, and after that at intervals of one or two months, according to the nature of

the case. In order that there may be no mistake, the mother or nurse should be given written directions. The treatment is continued until the visual acuity of the squinting eye becomes equal to that of the fixing eye, or until no further improvement can be got. Having employed this method for many years, and in a very large number of cases, I am able to state positively that there is no danger of its causing any permanent impairment of the power of accommodation.

This treatment will always prevent the deviating eye from becoming amblyopic. Its efficiency in curing amblyopia which has already been acquired will be greater the younger the child and the more recent the deviation. After about seven years of age usually not much improvement in vision can be obtained, though I have met with many exceptions to this rule.

Of course, carefully keeping the fixing eye tied up for not less than one-third of the child's waking hours would answer as well. But the child would need to be watched closely during the whole time, so that in any small household the mother and nurse would after a few weeks find the amount of supervision required too great a tax on their time and patience. But even the hard-worked mother of a large family, attending a hospital out-patient clinic, finds it no trouble to put a drop of atropine into the *fixing* eye *only* every morning, for as long as it may be required.

A disastrous practice, which until recently was invariably recommended in books, and is still employed by some surgeons, is that of ordering atropine for *both* eyes for children who are supposed to be too young for glasses. The object, of course, is to lessen the convergence by paralysing the accommodation. Atropine, used in this way, never brings about a permanent cure of the squint, though it occasionally causes a temporary suspension of the deviation. But the deviating eye is usually the more ametropic (this being generally the factor which decided in the first instance which should be the deviating eye), so that to paralyse its accommodation with atropine is the very way to ensure that this eye shall never under any circumstances be used. The most hopeless cases one sees, of blindness of the squinting eye, are those which have been treated with atropine for both eyes for a few months.

(4) Training the Fusion Sense.—In a case of unilateral or accidentally alternating squint, *if one sees the child early enough,* one should endeavour to remove the fundamental cause of the squint by training the fusion sense. For this purpose I use an instrument which I have called "amblyoscope." A description of the amblyoscope and of the method of training the fusion faculty will be found in the next chapter. The favourable time for fusion training is between the ages of three and five years. In children under

three years of age this treatment is apt to be rather difficult, though I have succeeded in many cases. After five years of age the fusion training takes longer, and a much less powerful "desire for binocular vision" is obtained. After six years of age it is seldom worth while to attempt fusion training at all. It is true that sometimes a patient who has squinted for many years may have binocular vision when his deviation is accurately corrected by operation at a much later date. But this small degree of fusion sense is not a fresh acquisition. He had it before he squinted, but it was too feeble to prevent the occurrence of a deviation, or even to cause diplopia.

Normally the fusion faculty begins to develop at a very early age, and, I think, reaches its full development by about the end of the sixth year. The education of the fusion faculty, at a time when this should normally be developing, is, in favourable cases, easily carried out and most satisfactory in its results. "Stereoscopic exercises," undertaken at a time when the child is old enough to take an intelligent interest in the process, are infinitely tedious and disappointing.

Of course, one should never omit fusion training in any suitable case which presents itself in private practice. But in a large hospital clinic it is physically impossible to find time for it except in a few selected cases for demonstration.

(5) OPERATION.—In cases of convergent squint

in which the deviation is not overcome by other means, operation becomes necessary. Two operative procedures may be employed either separately or in combination. They are tenotomy of the internal rectus muscle and advancement of the external rectus muscle; or the external rectus may be shortened, either by exsection of part of its tendon or by folding the tendon upon itself.

Tenotomy of the internal rectus consists in a division of the tendon of this muscle at its insertion into the globe. The eye then rotates outwards to an uncertain degree. This outward rotation usually tends to increase as time goes on. The eye also falls forward to a slight extent, so that the tenotomised eye is more prominent than its fellow and its palpebral fissure wider. A permanent weakness of inward rotation (adversion) of this eye is also produced.

In order to *advance* a muscle, the tendon is separated from the globe at its insertion. It is usually then shortened by removal of more or less of the tendon and muscle. The cut end of the muscle is then secured to the globe at a point further forward, nearer the cornea than its original insertion. By a properly performed advancement the eye is rotated to exactly the extent required. The results obtained by the accurate performance of the advancement operation described in chapter xii. are permanent, tending neither to increase nor to decrease with time. Both the

power and extent of the rotation of the eye in the direction of action of the advanced muscle are increased. The rotation of the eye in the opposite direction is not in any case weakened in force, though in extreme cases its extent may be slightly diminished. If the abnormal convergence does not exceed about 15°, its cure by advancement of one external rectus muscle causes no noticeable retraction of the globe. If the deviation be of higher degree than this, I advance both external recti.

The operations on the external ocular muscles are fully described and discussed in chapter xii.

The question may naturally be asked: "As the deviation in a case of convergent squint is not primarily due to a defect of the muscles, why should one seek to remedy this deviation by shortening a muscle?" The answer is this—In the presence of a defect of the fusion faculty, refractive error may cause a deviation, or the equilibrium of the convergence centre may, in some unknown way, be upset by a fright, blow on the head, whooping-cough, etc. If one is able to train the fusion sense *early,* the desire for binocular vision may overcome the deviation and produce a perfect cure. In many cases optical correction of refractive error causes the deviation to disappear. In other cases, as one cannot act directly upon the nervous centre which regulates convergence, one has to be content to act upon the peripheral motor organs. Take an illustration—One is driving a pair of horses. Sup-

pose the off-side horse has a habit of boring to the left. If any cause can be found (such as sore shoulder), one may cure the habit by removing this cause. If not, it is a reasonable proceeding to overcome his "deviation" to the left by shortening the off-side branch of the right rein.

Of the cases in which efficient treatment is carried out from soon after the first appearance of the deviation, only a small proportion ever need operation. But in some cases, without any observable defect in any single muscle, there is an imperfection in oculo-motor balance which, if the fusion sense had been perfect, would have given rise to an esophoria. Of the neglected cases, operation will be required in a much larger proportion.

The angle of the deviation is measured at each visit. If the measurements show a steady decrease, no operation is indicated. If they do not, operation will be required at some time or other.

Some squinters who have a little fusion sense, as well as many of those whose fusion sense has been educated by artificial means, are able to perceive a faint second image when their attention is especially drawn to it. Accurate operation here gives an ideal result, with binocular single vision. Inaccuracy, especially in a vertical direction, is apt to cause annoying and persistent diplopia; for a faint second image, which towards the periphery

of the field is only with difficulty perceived, may be very obtrusive when it falls near the macula.

In the case of older children and adults, if the eyes are straight while glasses are worn but convergent when they are removed, usually glasses will be worn constantly throughout life and no operation will be needed. But if the refractive error be only of moderate degree, I give the patient the option of operation which will enable him to dispense with glasses, except perhaps for reading. In the case of a boy whose proposed career might be unfavourably affected by the necessity of wearing glasses constantly, or of a lady whose appearance is a consideration, the choice has usually been for operation, and no patient has had cause to regret the decision. The operation is advancement of one external rectus or both. There is no tendency to divergence afterwards, whether glasses are worn or not. I have never performed tenotomy in such a case and believe that it would be especially dangerous.

If the angle is not higher than about 15° I advance one external rectus muscle. If the degree is higher than this I advance the external rectus of the other eye also, with an interval of ten days between the two operations, and dividing the effect equally between the two eyes. I do not now perform tenotomy of the rectus internus even in combination with advancement of the externus. I once used to do so occasionally. Many of these

patients have since suffered from insufficiency of convergence, and pain in the eyes in near vision. In some cases, in which tenotomy was followed by divergence, I have had to advance the tenotomised muscles.

One not infrequently sees a patient whose rectus internus has at some previous time been tenotomised, producing proptosis of the eye, but still leaving some degree of convergent squint. In this case an accurately performed advancement of the external rectus will cure the deviation, and at the same time draw the eye back into its proper position in the orbit.

Tenotomy is an unsatisfactory operation. The tendon is divided, and the result cannot in any individual case be foretold. Some cases turn out well, others do not. The effect produced by a tenotomy varies very widely in different cases. The effect may be insufficient or it may be about what is desired, or it may continue to increase for years, until a divergent squint is produced—a more hideous deformity than that which the operation was intended to cure.

The internal rectus should *never* be tenotomised in any case in which dynamic convergence is subnormal.

A patient requires very little care after the operation of tenotomy. This is a great advantage in a crowded hospital clinic. But I think tenotomy owes its popularity chiefly to the extreme facility

of its execution, almost no special knowledge being required. Accurate and certain performance of advancement, on the other hand, requires more skill and experience than any other operation in ophthalmic surgery.

Alternating convergent squint.—The treatment of an accidentally alternating squint is similar to that of a unilateral squint, except that there is no acquired amblyopia to be remedied. It must not be forgotten, however, that such a case, if neglected, may become unilateral, and that the deviating eye may then become amblyopic.

Essentially alternating squints are, fortunately, not very common. The treatment consists in optical correction of any ametropia which may be present, followed, in the majority of cases, by operation. Fusion training is impossible, as there is a total congenital absence of the faculty of acquiring fusion.

Occasional convergent squint.—The majority of occasional squints in young children are of the premonitory variety. Optical correction of any refractive error which may be present usually prevents the recurrence of the deviation, and so allows the natural development of the fusion sense to proceed.

Quite half the cases which are commonly supposed to be occasional squints, occurring in older children and adults, are really examples of esophoria (see chapter xi.).

If one whose fusion faculty is perfect suffers from anisometropia of so high a degree as to render the act of binocular vision difficult, he will be likely to manifest an occasional squint. Glasses should be ordered which give the sharpest vision in each eye, even though one eye be hypermetropic and the other myopic. The patient will soon become accustomed to the inequality in the size of the two images. An occasional squint may be caused by hypermetropia in a patient whose fusion sense is feeble.

Vertical deviation.—If operation be required in a case of true vertical deviation, this should consist in advancement of the inferior rectus muscle of the eye which turns up. The inferior rectus should never be tenotomised.

Apparent vertical deviation (p. 35) requires no treatment beyond that of the convergent squint.

CHAPTER VIII

THE METHOD OF TRAINING THE FUSION SENSE

FOR more than half a century attempts have, from time to time, been made to teach squinting patients to use both eyes together, by means of exercises with some form of stereoscope. Among the stereoscopes used for this purpose are Wheatstone's original instrument, Brewster's and Helmholtz's stereoscopes, Hering's haploscope, Holme's stereoscope, and, more recently, Javal's "Stéréoscope à cinq mouvements," Priestley Smith's "Heteroscope," Landolt's and Parinaud's stereoscopes, and very many other more or less similar instruments. Most of these are so arranged that they can be adapted to suit the angle of the patient's squint. But the proportion of cases in which they can be used is small, owing to the suppression of the vision of the patient's deviating eye. Javal attempted to overcome this suppression by prolonged occlusion of one or other eye, in the hope that, when at last both eyes were uncovered, the patient might have diplopia. All these instruments are intended for the use of patients who are old enough to follow intelligently the directions given them.

Probably most ophthalmic surgeons have, at some time or other, been in the habit of ordering "stereoscopic exercises" in cases of convergent squint, and have, after careful trial, given them up as useless. The reason of their failure is the very early age at which the fusion sense normally develops. Fusion training, to be of any material benefit, must be undertaken within this normal period of development.

Fusion training of young squinters under five years of age is, in suitable cases, quickly and easily accomplished, and the results obtained are most striking and gratifying. Between five and six the treatment is apt to take longer, and the results to be less perfect. After the age of six or, at the latest, seven years, the results are seldom worth the time and trouble which they cost.

There are two great difficulties in the way of fusion training in the case of young children :—

(1) Though the visual acuity of the child's deviating eye may perhaps be perfect, the vision of this eye is suppressed, so that he is ordinarily unable to receive impressions from it except when the other eye is closed.

(2) The child is far too young to understand the purpose of fusion training or to follow the directions of the surgeon. He will, therefore, only permit the exercises so long as he finds them attractive and interesting.

After many experiments, I devised an instrument

with the help of which I have to a great extent succeeded in overcoming these difficulties. I have called it "amblyoscope"—an instrument by means of which a non-seeing eye is trained to take its share in vision. The amblyoscope has retained its present form since 1895.

The Amblyoscope.[1]—The instrument consists of two halves joined together by a hinge. Each half consists of a very short brass tube joined to a longer tube at an angle of 120°. At the angle of junction of the tubes is an oval mirror,[2] protected on the outside by an oval plate of brass. Each half of the instrument has at its distal end an object-slide carrier, and at its proximal end a convex lens having a focal length of five inches—the distance of the reflected image of the object-slide. In front of each lens is a slot into which a prism, axis vertical, may be inserted if required. The diameter of the tubes is $1\frac{1}{2}$ inches.

A brass arc[3] connects the two parts of the

[1] The amblyoscope is made by Mr. Hawes, optician, 79, Leadenhall Street, London, E.C., and by the principal manufacturing opticians in England and abroad.

[2] The mirrors must be extremely thin. If they are merely pieces of ordinary thick mirror glass there will be blurring, owing to reflection from the surface of the glass as well as from that of the mercury. Mirrors silvered on the surface answer well but are easily scratched. I have tried polished speculum metal. This is satisfactory but very expensive. The mirrors must be accurately set perpendicular to the plane of the tubes.

[3] In the earlier forms of the instrument I had the arc marked in degrees, as I thought that the instrument might

instrument, being clamped on one side by a binding screw set in a long slot and on the other by a binding screw set in a short slot. When the screw in the long slot is loosened, the two parts of the instrument can be brought together to suit a convergence of the visual axes up to 60°, or separated to suit a divergence of as much as 20°. When this screw is tightened and the screw in the short slot

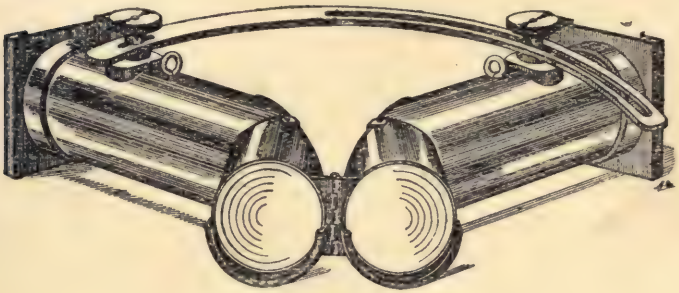

FIG. 12.

loosened, an amplitude of movement of about 10° only is permitted.

The convex lenses of course render unnecessary any adjustment of the instrument for the patient's inter-pupillary width.

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also be used for the subjective measurement of heterophorias. I found, however, that it was of no use for this purpose. No instrument, in which the objects looked at are near the eyes, is reliable for measuring heterophoria. Though the lenses render accommodation unnecessary, the patient unconsciously accommodates for an object which he knows is near.

Illumination of the object-slides.—Each object-slide is illuminated by a separate electric lamp.

A stout brass rod, about two feet in length, is held in brass sockets at each end. The brass sockets are screwed to a board, which is firmly secured to the wall of the consulting-room in such

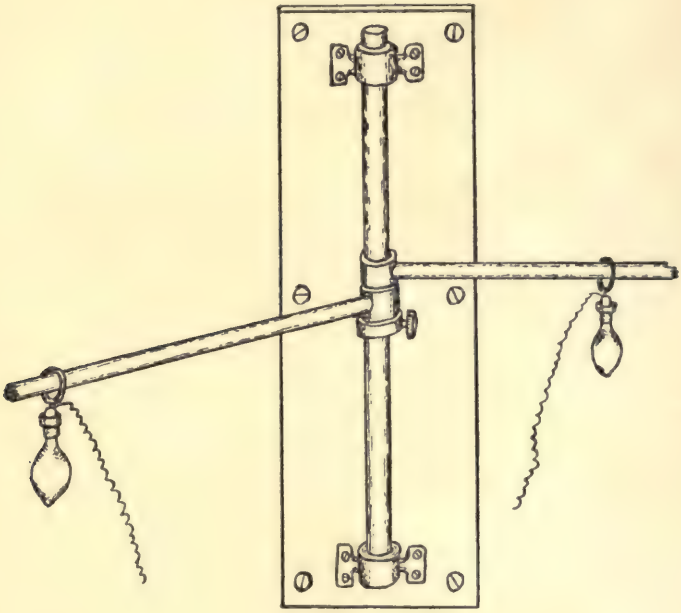

FIG. 13.

a position that the brass rod is vertical. A brass collar slides up and down the vertical rod, and is fixed by a thumb-screw at the height required. This collar supports two independent collars, each of which has attached to it a long horizontal arm. Each of these arms is four feet in length, and is

free to move in a horizontal plane independently of the other. An electric lamp is suspended under each horizontal arm from a ring which slides along the arm. The illumination of either of the object-slides in the amblyoscope may be separately increased or diminished by bringing its lamp nearer or pushing it further away.

Two dark-room bracket lamps serve equally well. Or a single lamp in a partially darkened room, or daylight from between half-drawn curtains could be used in most cases.

The object-slides.—Fig. 14 shows the familiar vertical slits with their control marks. They are not used for fusion training; one could not induce a young child to study such uninteresting objects. They are shown because they are useful in making experiments with older persons.

The devices used in fusion training are of three classes:—

(1) Those which do not require any blending of images, but only simultaneous vision of dissimilar objects with the two eyes. Fig. 16, showing a cage on one slide and a bird on the other, is an example. Other pairs of devices of this class are a clown and a hoop, a mouse and a trap, a clock-face and hands, etc.

(2) Devices of the second class, of which figs. 17 and 18 are examples, require true fusion of images in order that the full picture may be seen. The pictures on each pair of slides are exactly similar,

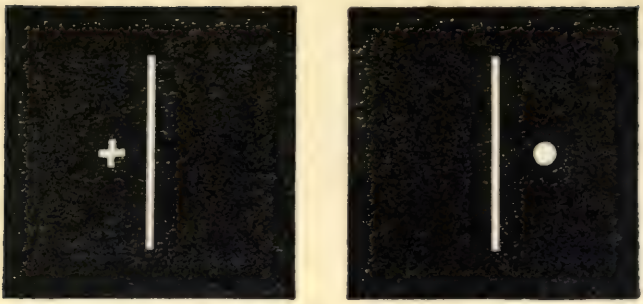

FIG. 14.

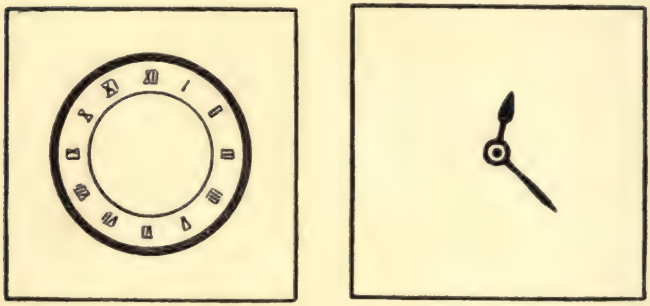

FIG. 15.

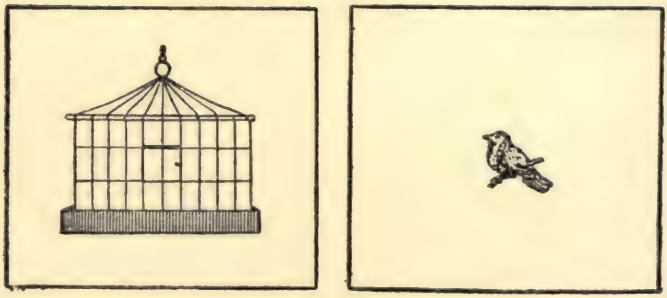

FIG. 16.

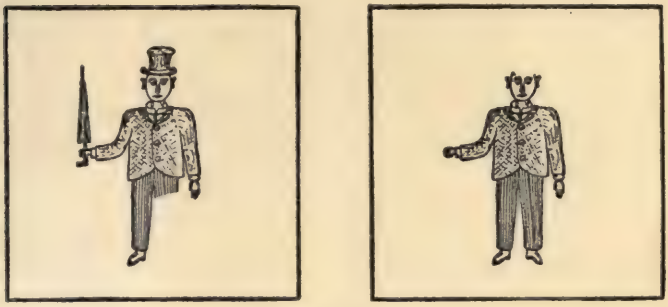

FIG. 17.

FIG. 18.

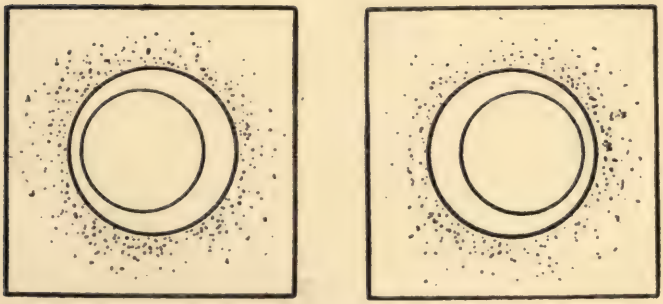

FIG. 19.

except that part of the design is omitted in one and a different part in another. For example, in fig. 17 a leg is omitted in one slide and the hat in the other. A child who blends the images sees a man with two legs and a hat.

(3) Devices such as fig. 19 can only be appreciated by patients who have the third grade of binocular vision, the sense of perspective.

The designs are drawn on 1½-inch squares of thin translucent paper. The paper is then pasted on squares of glass. There is no difficulty in making designs of the first class. Object-slides of the second class are made as follows: Draw, on pieces of paper 1½ inches square, rough, simple pictures, such as a horse, a clown, a cow, a man with a hat and pipe, etc.; the more grotesque the better the children seem to like them. Then make two tracings of each on the translucent paper, omitting a different part of the picture in each tracing.[1]

Fusion Training.—The education of the fusion sense should be undertaken at the earliest possible age. I repeat this because it is of extreme impor-

[1] "Stereoscopic views" are of no practical use whatever for fusion training. But I once had made some tiny transparent stereoscopic photographs to fit the amblyoscope. My idea was that a child who wearied of my own artistic efforts might be shown some of these views, occasionally, by way of a change. But I found that young children took no interest in them. They much preferred simple pictures which they could understand.

tance—the only key to success. In the case of a child of average intelligence, it is quite easy to use the amblyoscope at the age of three to three and a half years. I have succeeded in many cases before three years of age, but it is often rather difficult to keep the attention of these very young children.

The deviating eye must not be too blind. In cases which have received *efficient* treatment since soon after the first appearance of the deviation, the vision of each eye is nearly always perfect. But, in neglected or inefficiently treated cases, the deviating eye is often very blind. (Compare tables iii. and v., pp. 76 and 77.) I do not as a rule attempt fusion training in a child who, after all possible means have been employed to restore the sight of the deviating eye, is still unable to see the 1-inch ivory ball at six yards.

A squinter sees with his fixing eye only, or, under certain circumstances, with the deviating eye only, but not with both simultaneously. The first step in the treatment is to overcome this suppression. If the child has any refractive error this is corrected by spectacles. It occasionally happens that the mirror test (p. 80) has revealed a true vertical deviation. In this case corresponding prisms, axes vertical, are inserted in the slots in front of the lenses. Seated in a chair facing the lighting apparatus, one takes the child on one's knee and adapts the amblyoscope roughly to the angle of

his squint. One puts in the instrument first a pair of object-slides, which require no fusion, only simultaneous perception—those shown in fig. 16, for example. Each light is at first about four feet away from its object-slide. Suppose the cage is before the fixing eye and the bird before the squinting eye. The child, on looking into the amblyoscope, will see only the cage. He is told to look for the bird, while the light before the deviating eye is brought nearer and nearer. At last a point is reached when the illumination of the object-slide before the deviating eye becomes so intense that the vision of this eye can no longer be suppressed. The child suddenly says he sees the bird he was told to look for. But he has now usually lost sight of the cage before the fixing eye. The relative distances of the lights are then readjusted until, after a few minutes of alternation of the vision of the two eyes, the child sees the bird and the cage simultaneously. The child is then allowed to grasp the amblyoscope with both hands, while the surgeon, putting his hands over the child's, converges and diverges the two halves of the instrument, in order to make the bird appear to go in and out of the cage. Other similar pairs of object-slides are then shown. The child soon learns to move the instrument himself, so as to put the bird in the cage, the cat on the chair, the clown in the hoop, etc. One must talk to the child all this time,

as it is only by encouraging him to chatter that one learns what he really sees.

A pair of slides requiring fusion of images is now shown—fig. 17, for example. The child at first sees two men, each picture being imperfect. Soon a position is found in which the child sees one man having two legs and a hat. The binding screw in the long slot is now tightened and that in the short slot loosened, so that the amplitude of movement between the two halves of the amblyoscope is restricted to about 10°. Other pairs of slides requiring fusion of images are successively shown the child, and he is encouraged by one's remarks to examine every part of the fused picture. After a time it is found that the angle of convergence of the instrument may be varied slightly without the fused picture coming to pieces. The child has now, under these special conditions of illumination and convergence, the second grade of binocular vision —true fusion with some amplitude.

The next step is to increase the amplitude of fusion. The intensities of the lights and the angle of convergence of the amblyoscope are arranged as before. Devices requiring fusion, such as figs. 17 and 18, are shown. An attempt is made gradually to diverge and converge the two halves of the instrument, more and more, while the child is examining and talking about the various pictures shown him. After some practice, in the case

of a young child, a considerable range of movement becomes possible, fusion being still maintained. This "amplitude of fusion" may, for practical purposes, be taken as a measure of the extent to which the fusion faculty has been developed.

A child who has any considerable amplitude of fusion will nearly always be found to have acquired the third grade of binocular vision also—the sense of perspective. The slides, fig. 19, are shown, and the child is asked whether he is looking at the outside or the inside of the tub. He will at once say "the inside." If these slides are now changed from one tube to the other, he will see the tub bottom up.

The child having now acquired the highest grade of binocular vision under these special conditions of illumination and convergence, the next step is gradually to equalise the light before the two eyes. This may readily be done, at this stage, without a return of the suppression.

If the child be young enough, a fairly strong "desire for fusion" may usually be created in five or six lessons, given at intervals of one week. If the training can be efficiently carried out at home, the child should have a lesson of a few minutes' duration every morning.

It must be remembered that the prime object of these exercises is the training of the fusion sense at a time when this may be successfully

accomplished — not a mere remedying of the deviation. In many cases, however, the "desire for fusion" thus established directly brings about a cure of the squint. In the larger group of cases, in which optical correction of refractive error is depended upon to lessen, and perhaps overcome, the deviation, there is no danger of the newly acquired faculty of fusion being lost meanwhile. If even a faint degree of fusion sense has once been acquired, its persistence is truly remarkable. No matter how many years binocular vision may be in abeyance, it seems as difficult to forget as the art of swimming. If the periodic measurements of the angle of the deviation show that this is not decreasing under optical treatment, advancement of one external rectus, or of both, should be performed. The mechanical obstacle to fusion having been removed, the child's trained fusion sense then finds its expression in the act of binocular vision.

Fusion training, in the case of these young children, must, in the first instance, be carried out by the surgeon himself. It is of no use giving the amblyoscope, or any other instrument, to the mother for use at home until she has been instructed in the method of using it. An intelligent mother can carry out the training quite satisfactorily. The whole process is very simple, but requires infinite patience.

In this description of fusion training I wish to

draw attention rather to the principles than to the instruments by the help of which they have been defined and applied. Though these methods have been constantly employed for many years, it is possible that they may be improved upon ; but the principles are unalterable.

CHAPTER IX

DIVERGENT SQUINT

COMITANT divergent squint presents two distinct varieties differing widely in their pathology, appropriate treatment, and prognosis. They may be called, respectively, myopic and neuropathic.

MYOPIC DIVERGENT SQUINT.

This is much less common than it was twenty years ago, owing to the greater attention which is now paid to the eyes of school-children. The divergence most frequently first makes its appearance at about ten or twelve years of age. It may be either unilateral or alternating, more commonly the former. Usually the patient is known to have been short-sighted for four or five years, and the short-sightedness has been increasing, until, at the time of the appearance of the divergence, there is myopia of perhaps 5 or 6 D or more. As a rule the fusion sense is well developed, though I have sometimes found it deficient. The deviation is usually not constant; at one moment the eyes may be "straight" and the patient have binocular vision; a few minutes later one or other eye may exhibit

a high degree of divergence. Myopic divergent squints of low degree are rare.

From observing and carefully questioning many of the more intelligent patients, I conclude that the usual mode of origin of a myopic divergent squint is as follows: Soon after the child begins school it is found that he is not easily able to see the black-board. He is given a front seat where he can see, and nothing more is done. A few years later, say at twelve or thirteen years of age, the myopia has increased so as to bring his far point very near his eyes. All distant objects are blurred. In reading, he has to hold his book so near his eyes that it becomes very difficult to converge steadily to the required extent. He complains that "the words run into each other," and he soon tires. One frequently hears that, at about this time, he acquired the habit of holding his head very much on one side in reading. In any case, he gives up the struggle and allows one eye to wander outwards, while he reads without effort with the other. At first there is no actual divergence—only a failure to converge while the other eye is engaged in near vision. But, as a result of convergence not being used, the function becomes weakened, so that either eye diverges when screened or when the other is being used in near vision. When the myopic eye has become divergent there is no diplopia in reading, even though the fusion faculty be perfect, because

the divergent eye is directed towards the misty distance. When the patient looks up from his book, the divergent eye at first always recovers itself. After a time, as the habit becomes confirmed, this eye often remains widely divergent in distant vision. The picture of distant objects formed in the fixing eye is already blurred and indistinct, so the very faint eccentrically placed image formed in the divergent eye causes no tendency to fusion. But when the divergent eye partially recovers its normal direction, so that it receives a more centrally placed image of the object to which the fixing eye is directed, the desire to blend these two blurred images causes the eyes to become straight. This probably explains why in a case of myopic divergent squint the eyes are sometimes straight, sometimes widely divergent, but scarcely ever divergent to a slight degree.

The *treatment* of a case of myopic divergent squint consists in exactly correcting the myopia and myopic astigmatism. The patient should wear the glasses always, both for near and distant vision. Patients sometimes at first complain that the glasses make print appear small. Children soon become accustomed to them. But high myopes who first begin to wear glasses in adult life, in some cases, require a second pair of glasses for near vision, about 1 D weaker than the distance glasses. Their power of accommodation

has become feeble for want of exercise. Glasses with which the patient can see distinctly usually cause a rapid disappearance of the squint in fairly recent cases, and frequently even in cases which have lasted for many years. But it often happens that the prolonged disuse of the function of dynamic convergence weakens the static convergence, so that this becomes a negative quantity, and a tendency to divergence remains. While both eyes are open and the glasses are worn, the fusion faculty prevents any deviation, but if one eye be shaded for a moment it may diverge and remain divergent for a second or two after the removal of the shade. Operation is rarely necessary. But in some cases, usually of long standing, in which the static convergence remains very deficient, advancement of an internal rectus adds greatly to the patient's comfort.

It must not be forgotten that the case may be complicated by muscular exophoria.

Infantile myopic divergent squint, excluding cases of buphthalmos, is very rare, for a myopic infant, if he squints at all, usually squints inwards. I have notes of a few cases. They differed from unilateral convergent squints only in the kind of refractive error and the direction of the deviation. The treatment was similar to that employed in convergent squint.

Neuropathic Divergent Squint.

There is a well-defined and fairly large group of cases which I have named "neuropathic divergent squint." The term may be open to objection, but, as these cases had never before been investigated and described, it was necessary to give them a name, and this seemed more appropriate than anything else I could think of.

The divergence nearly always dates from infancy. It may be constant or occasional, unilateral or alternating. In the constant cases there is a total absence of the fusion sense. In the occasional cases there is a feeble degree of binocular vision when the eyes are "straight"; when an eye diverges there is usually no diplopia. The refraction, as a rule, is normal. In the constant unilateral cases there may be acquired amblyopia. In other cases the vision of the eye is perfect. The divergence varies greatly in degree, even in a case in which it is constantly present. This is due, not to any variation in the motor balance of the eyes in the position of rest, but to the varying effort of dynamic convergence by which the patient unconsciously endeavours to correct the faulty relative positions of the eyes. The power of dynamic convergence is always deficient and varies from time to time in an extraordinary manner. A weak faculty is apt to be irregular in its action, just as a surgeon in ill-health cannot

so accurately perform a delicate operation. In these neuropathic divergent squints, the association between accommodation and convergence is usually very slight. One not infrequently sees a patient who can nearly always voluntarily correct the faulty position of his divergent eye without much effort, but who habitually allows this eye to diverge while he is exercising say 3 or 4 D of accommodation in near vision. In young subjects the power of rotation of each eye separately is usually normal in every direction. In long-standing cases, however, the power of independent inward rotation is deficient.

The subjects of neuropathic divergence are generally bright, quick-witted, intelligent, and they are nearly always very "nervous" and highly strung. One frequently sees the same defect in other members of the family. A family history of epilepsy or insanity is very common. I find neuropathic divergence more common in females than in males, in the proportion of about 7 to 2.

In addition to the adverse influence which the deformity may exert upon a boy's career or a girl's prospect of marriage, the consciousness of it often seems to produce a peculiar mental attitude in these sensitive children—a feeling of isolation and of being somehow different from other children. Frequently there is frontal headache and aching in the eyes.

Treatment.—There is seldom any notable refractive error, and when there is its correction produces no effect upon the divergence. Attempts at fusion training nearly always fail, however early the patient may be seen. One's only recourse is operation. A shade should be worn over one or other eye for several weeks, so that the eye may settle into its position of rest, and one can see exactly how much is to be done. Then both internal recti should be advanced (*e.g.*, Case B, 166, p. 165). The externi should never be tenotomised. An accurate operation will cure the deformity permanently. Cases in which divergence is not constantly present and in which there is some slight degree of fusion are especially favourable (*e.g.*, Case B, 165, p. 164).

Non-comitant Divergent Squints, other than Paralytic.

Divergence in extreme myopia.—In cases of progressive myopia of very high degree it is not uncommon to find the visual axes divergent. These cases differ entirely from ordinary myopic divergent squints. They are non-comitant. The arcs of rotation of each eye are sub-normal in every direction. The divergence begins imperceptibly, and increases slowly up to about 20°. It is generally held that the divergence is caused mechanically, by the egg-shaped eyes adapting

their long axes to the divergent positions of the orbits. I believe this explanation to be correct. Increased tension of the external recti does not appear to be an important factor, as I have, in three such cases, seen both these muscles tenotomised without result. Operation in these cases is not advisable.

Divergence of blind eyes.—When both eyes are blind they almost invariably diverge. When one eye only is blind, its behaviour will depend, to a great extent, upon the state of the refraction of the seeing eye. If this is normal or myopic the blind eye will, as a rule, diverge; if it is markedly hypermetropic it will usually converge.

If the refraction of one eye be normal, or nearly so, while the other eye is very highly myopic, the latter is, in the absence of optical correction, practically a blind eye, and will behave as such. The same remark applies to an eye which has become very amblyopic from disuse.

Divergence secondary to tenotomy of an internal rectus muscle.—As already explained, in a large proportion of cases of convergent squint with hypermetropia the constant wearing of glasses gradually causes the visual axes to become less convergent. It would obviously be dangerous to tenotomise an internal rectus while this change is in progress.

Even if tenotomy be performed only in cases in which optical treatment has failed to overcome the

convergence, the risk of divergence is considerable. In tenotomy the tendon is divided, an attempt usually being made to spare its lateral expansions. Usually the cut end of the tendon becomes connected to its old insertion by an irregular band of scar tissue, which may or may not subsequently stretch. Tenotomy is called a "setting back" of the tendon, because it is supposed that the tendon becomes directly reattached to the globe further back. I believe that this seldom happens after a neatly performed tenotomy. Sometimes the tendon fails to become reattached to the globe at all. In this case a surgeon who attempts to readvance the tenotomised muscle may fail to find it unless he knows where to look for it. Its anterior end will be found behind the sunken caruncle, lying against the inner wall of the orbit (see p. 226).

Divergence caused by tenotomy nearly always slowly but surely increases, until the divergent and bulging eye becomes a very unpleasant disfigurement. When the divergence has become considerable there is constant aching pain in the eye and behind the eye.

Secondary advancement in such cases has more often than not resulted in complete cure of deformity and pain, and in the worst cases a great improvement has been produced. It is surprising how little atrophied one often finds the tenotomised muscle, even when the eye has

been divergent for years. Though it is advisable to deal with the case as early as possible, lapse of time is a less important factor than the degree of damage which has been done by the tenotomist.

Consecutive Divergence.

I have records of some rather rare cases in which, in the course of years, convergent squint has given place to divergence, without any operation having been performed. The eyes were hypermetropic and remained so throughout. In all these cases the change was gradual and continuous; *there was no period at which the angle of the convergence was stationary*. These cases serve to emphasise the importance of taking measurements at each visit.

CHAPTER X

THE TREATMENT OF SQUINT—ILLUSTRATIVE CASES

THE following clinical notes, copied from my case-books, will serve to explain further the methods of treatment, and to show the results which may be expected therefrom. The cases were not the most favourable, nor the most difficult, but are selected as being instructive. Of course, the different examples are not quoted in the proportion in which they are met with in practice, as this would involve useless repetition of the more common cases.

As a rule, it is only possible to get a real cure, with perfect sight in each eye, in a case in which efficient treatment is commenced early. But, unfortunately, one sees a considerable proportion of the cases for the first time after years of neglect, or perhaps inadequate or even harmful treatment. I have, therefore, also given some examples of these old cases.

CASE B, 23. *February* 4, 1896.—A boy, aged 2 years 11 months, suffering from convergent squint. He had squinted for about ten or twelve months. L. E. was convergent 28°. Fixation was present in

L. E., but unsteady, and the sight of this eye had deteriorated so much that, when R. E. was bandaged, he could not find a white-handled penknife on the floor, unless it was close to his feet. Abversion L. E. was perfect. Ordered, atropine ointment, 1 per cent., thrice daily for both eyes, for retinoscopy.

February 11.—Convergent squint, L. E. 32° with atropine. Retinoscopy—each eye + 2·75 D sph. + 0·5 D cyl. ax. vert. Ordered spectacles + 2·25 D sph. + 0·5 D cyl. ax. vert. R. E. to be occluded for one week, then guttæ atropinæ 1 per cent. for R. E. *only* every morning.

March 3.—Child uses R. E. (atropised) in distant vision, and L. E. (unatropised) in near vision. Convergent squint L. E. 20°, with glasses. Ordered, continue.

April 9.—Similar note. Convergent squint L. E. 17°. Ordered, continue.

May 29.—Child now uses (unatropised) L. E. and turns in (atropised) R. E. always, both in near and distant vision. Ordered, stop the drops, and come again in one month.

June 18.—Squint nearly alternating, slight preference for squinting with L. E. Convergent squint L. E. 16°. Fusion training. Ordered, atropine for R. E. only, every morning for a month, then stop it and bring the child to me two or three weeks later.

August 4.—Fusion training with amblyoscope. Child sees both images readily and can sometimes blend them.

August 7.—Fusion training.

August 10.—Fusion training. Child blends images readily.

August 14.—Fusion training. Child has considerable amplitude of fusion.

August 18.—Fusion training.

August 25.—No squint with glasses. The mother says that the eyes have been straight since the last

lesson, though he has turned the eye in, occasionally, for an instant.

November 6.—Child never squints now with glasses, though he occasionally does so without them, the nurse says. No squint seen now either with or without glasses.

October 8, 1897.—Child going on well. He never squints now.

September 29, 1898.—Spectacles too small. Ordered, repeat them.

June 13, 1901.—(Aged 8 years 3 months.) The patient looks over his glasses. He reads $\frac{6}{6}$, each eye, without them. Ordered, atropine for repetition of the retinoscopy.

June 20.—Retinoscopy each eye + 2·25 D sph. + 0·5 D cyl. ax. vert. Vision, each eye $\frac{6}{6}$. Ordered, spectacles 0·5 D lower than the retinoscopy, with permission to take them off while playing games.

August 19, 1902.—The boy has only used his glasses for school work since last visit, and never when out of doors. He has not been seen to squint for years.

Soon after this, the boy entirely gave up wearing glasses. Subsequently he passed into the Royal Navy as a cadet, and is now a sub-lieutenant. It need scarcely be said that, if efficient steps had not been taken to restore the sight of the left eye before it was too late, this career would have been barred to him.

Case B, 18. *November* 7, 1895.—A girl, aged 2 years 1 month, was brought to me, suffering from C. S. R. E. 33°. She had squinted since she had whooping-cough aged 1 year 4 months. She had central fixation in R. E., but she evidently did not see well with it when the L. E. was tied up. Abversion perfect. Bright, intelligent girl, only child. Her mother has a convergent and amblyopic right eye. Ordered, atropine for retinoscopy.

November 13.—Retinoscopy R. E. + 1·75 D sph. L. E. + 1·25 D sph. C. S. R. E. 26°, with atropine. Ordered, spectacles 0·25 D less than retinoscopy; also guttæ atropinæ 1 per cent., one drop in L. E. *only*, every morning.

December 11.—Child uses R. E. (unatropised) in near vision, and L. E. (atropised) in distant vision. Ordered, continue.

February 6, 1896.—Same. Ordered, continue.

April 2.—Child uses R. E. (unatropised) now, both in near and distant vision, and squints with atropised L. E. C. S. L. E. 27°. Ordered, stop atropine.

June 4.—Squint alternates 22°. Fusion training with amblyoscope: child readily sees both images, but I cannot be certain that she blends them.

June 10.—Fusion training: child learned to blend images quite easily. Fusion training continued until—

July 7.—Child now has a considerable amplitude of fusion, C. S. alternating 23°. As the fusion faculty is well developed and the deviation is stationary, I advised operation, especially as, in view of the insignificance of the refractive error, spectacles can be dispensed with. Fusion training was continued until—

October 5 (aged 3 years).—C. S. alternating 22°. I advanced the right external rectus muscle, under chloroform. The method employed was in all essential particulars that described in chapter xii.

[NOTE.—I do not usually operate so early as 3 years of age, owing to the difficulty of keeping the little patient quite still in bed afterwards.]

November 3.—R. E. is not opened quite so widely as L. E., and there is still some redness of the conjunctiva. There is no squint now, and the child has perfect binocular vision.

No glasses required. I saw the little girl in January, 1897, October, 1897, June, 1899, and again on

November 12, 1902.—The child is now nine years old. She has $\frac{6}{6}$ vision, each eye, and perfect binocular vision.

December 16, 1914. (Eighteen years after the operation.)—At my request the patient, who is now married, came to see me. She has perfect sight in each eye, and perfect binocular vision. All movements of the eyes are normal. That there was ever anything amiss with her eyes is, to her, nothing more than a family tradition.

CASE B, 192. *November* 20, 1899.—A lady, aged 23 years. *History.*—She began to squint with the L. E. when she was about 1 year old. She was treated from time to time with "drops." At about 4 years of age she was ordered glasses. She has worn glasses ever since. When she was 8 years old the L. E. was operated upon (tenotomy).

She now has convergent squint L. E. 16° both with and without her glasses. Vision of R. E., with glasses, is $\frac{6}{6}$. L. E. has lost central fixation, and with it she can just count fingers close to her face. As the result of the tenotomy, the L. E. is prominent and the caruncle sunken. Retinoscopy under homatropine—R. E. + 1 D cyl. ax. vert., L. E. + 1·75 D cyl. ax. 70° down and out, at approximate macula. The blindness of the left eye is now, of course, quite incurable. She wishes to have something done to remove the deformity.

December 5.—I advanced the left external rectus muscle, under cocaine (by the method described in chapter xii.).

December 20.—There is a little redness remaining from the operation. The eyes are quite "straight," and the advancement has had the effect of replacing the L. E. in its proper position in the orbit. Ordered, rigid pince-nez + 1 D cyl. ax. vert.

June 14, 1901.—Eyes quite normal in appearance, both with and without the glasses.

November 23, 1904.—Eyes normal in appearance: no discomfort.

Since then I have, unfortunately, lost sight of this patient.

CASE D, 318. *April* 13, 1900. Boy, aged 1 year 5 months, seen at Moorfields Hospital. The mother said he had measles in February and he "has squinted only a few weeks." C. S. R. E. 37°. Steady central fixation R. E. Ordered, atropine, for retinoscopy.

April 24.—C. S. R. E. 22° under atropine. Retinoscopy—R. E. + 5·5 D sph. L. E. + 4·5 D sph. Ordered, spectacles R. E. + 5 D L. E. + 4 D. Guttæ atropinæ 1 per cent. to be put in L. E. *only* every morning.

May 8.—Child uses (unatropised) R. E. and squints (atropised) L. E. always. Ordered, stop drops.

June 5.—Squint alternates, 21°.

August 7.—No squint while wearing glasses. C. S. R. E. about 20° without glasses.

September 10, 1901.—Glasses too small now, repeat them.

November 7, 1902.—No squint seen here. He is said to turn in R. E. sometimes when his glasses are taken off at bedtime.

March 7, 1905.—No squint with glasses. Occasional squint, with diplopia, when glasses are removed.

[In this case, it would appear that the fusion sense was late in developing, so that at the age of about 15 months hypermetropia caused a squint. Correction of hypermetropia brought about a gradual relaxation of the abnormal convergence, which permitted the natural development of a useful, but never powerful, fusion sense.]

CASE A, 489. *May* 10, 1900.—Boy, aged 3 years 4 months, seen at the West Ham Hospital. C. S. L. E. 34°. Fixation L. E. was lost. Abversion L. E. full. The mother said he had "squinted on and off for about a year." Ordered, atropine for retinoscopy.

May 17.—C. S. L. E. 27° with atropine. Retinoscopy—R. E. + 3·75 D sph. + 0·75 D cyl. ax. 25° down and out. L. E. approximately the same. Ordered, spectacles for constant wear, R. E. + 3·5 D sph. + 0·75 D cyl. ax. 25° down and out. L. E. + 3·5 D sph. *pro tem.:* also R. E. to be occluded by a gauze pad secured by strapping-plaster, for one month.

June 14.—Steady central fixation L. E. Ordered, guttæ atropinæ 1 per cent. for R. E. *only* every morning for one month.

July 12.—Child now uses (atropised) R. E. in distant vision, and turns in L. E. In near vision he uses (unatropised) L. E., and turns in R. E. C. S. L. E. 23°. Ordered, continue.

August 30.—Child uses R. E. distant vision and L. E. near vision. When glasses are taken off, he always fixes with L. E. and turns in the (atropised) R. E. Ordered, continue.

October 25.—Child uses (unatropised) L. E. and turns in the (atropised) R. E. always, both with and without the glasses. Ordered, stop the atropine and return in one month.

December 6.—C. S. L. E. 10°. The child uses L. E. almost as readily as R. E. Fusion training every week until—

January 24.—Fusion training. Images blended at once. A fair amplitude of fusion.

February 7.—No squint with glasses.

July 11.—No squint with glasses. When glasses are taken off the child has no squint, as a rule; but when he is told to look at a picture L. E. turns in about 35° to 40°, and he says he sees two books (he volunteered this statement without any questioning).

May 29, 1902.—Child never squints now. Ordered, atropine for both eyes for repetition of the retinoscopy.

June 7.—Retinoscopy, R. E. + 3·5 D sph. + 0·75 D cyl. ax. 30° down and out. L. E. + 3·5 D sph. + 1 D cyl. ax. 30° down and out. Vision with these

glasses R. E. $\frac{6}{9}$, L. E. $\frac{6}{9}$. Ordered, spectacles 0·5 D less than the retinoscopy.

CASE D, 832. *May* 6, 1902.—A boy, aged $9\frac{1}{2}$ years, was brought to the Royal London Ophthalmic Hospital because he could not see with the right eye. The father said that the boy squinted when he was teething. He was taken to an eye hospital, where he was given ointment (probably atropine) for both eyes for about a year. At about $3\frac{1}{2}$ years of age he was given glasses. The glasses gradually "cured the squint, but the sight of the eye which used to squint is almost gone."

The boy is wearing + 4 D sph. each eye. Vision with his glasses—R. E. counts fingers at two feet, L. E. $\frac{6}{9}$. Fixation R. E. is, of course, lost. He has C. S. R. E. 4°, with his glasses. Abversion R. E. full. Ordered, atropine for retinoscopy.

May 9.—Retinoscopy L. E. + 4 D sph. + 1 D cyl. ax. vert. R. E. approximately the same. Vision with glasses—R. E. not improved, L. E. $\frac{6}{9}$. Ordered, spectacles 0·5 D less than retinoscopy. The blindness of the R. E. is now of so long standing that it would be quite hopeless to attempt to restore the sight.

CASE B, 3. *May* 22, 1893.—A girl, aged 1 year 5 months, daughter of one of my former fellow-students. She had C. S. R. E. about 35°. She had squinted for six or seven months. R. E. has not lost the power of central fixation. Ordered, atropine for retinoscopy.

May 25.—Retinoscopy each eye + 4 D sph. Ordered, glasses each eye + 3·5 D sph.: also, guttæ atropinæ 1 per cent. L. E. *only* every morning.

June 26, 1893.—Glasses worn well. When the child is induced to look at anything close to the eyes, she uses the R. E. (unatropised) and turns in the (atropised) L. E. At other times she has C. S. R. E. about 30°. Ordered, continue drops for L. E. *only*.

July 28.—Child uses L. E. (atropised) in distant vision and R. E. (unatropised) in near vision. When glasses are taken off, she always uses R. E. and squints with (atropised) L. E. Ordered, continue.

September 5.—Child always uses R. E. and squints with the (atropised) L. E. both in near and distant vision. Ordered, stop the atropine.

April 23, 1894.—C. S. nearly alternating. More often fixes with the R. E., which was at first the squinting eye. C. S. 21°.

November 6, 1895.—Owing to my absence abroad the child was left much longer than she should have been. C. S. L. E. 13°. Ordered, atropine for repetition of retinoscopy.

November 13.—Retinoscopy + 3·5 D sph. + 0·5 D cyl. ax. vert. each eye. Ordered, glasses 0·5 D less than the retinoscopy. As the child now squinted constantly with L. E. (the originally fixing eye) I ordered atropine to be put in the R. E. *only* every morning.

November 27.—She uses R. E. (atropised) in distant vision and L. E. (unatropised) in near vision. Ordered, continue.

December 20.—Child now uses L. E. almost always in preference to the (atropised) R. E. Ordered, stop atropine.

January 15, 1896.—C. S. nearly alternating 11°. Fusion training.

January 20.—Fusion training.

January 27.—Fusion training. Child blends images.

February 4.—Fusion training. Child has some amplitude of fusion.

February 13.—Fusion training. Considerable amplitude of fusion.

February 20.—Child has frequently said that she sees two faces, etc., since last visit. Fusion training.

February 27.—No squint.

June 5.—No squint with glasses. C. S., with diplopia, when glasses are taken off. Diplopia is so intense that the little girl remarks it at once.

October 19, 1898.—Glasses too small. Ordered, repeat the glasses with larger frames. The child is never seen to squint now. Vision R. E. $\frac{6}{6}$, L. E. (the originally fixing eye) $\frac{6}{6}$ easily, $\frac{6}{6}$ partly.

October 24, 1902.—(Age now 9 years 10 months.) She has grade iii. binocular vision, with a good amplitude of fusion. She never squints now, either with or without her glasses. She has hitherto worn her glasses always, but I have given her permission to dispense with them during her dancing lessons and gymnasium practice.

March 4, 1903.—Since the last visit the patient has seldom worn her glasses except for school work. Without glasses the eyes have sometimes been straight, but more often strongly convergent, with faint diplopia. The girl and her parents desire operation with a view to her leaving off glasses except for reading, etc. Owing to family reasons this had to be postponed until the autumn. Glasses to be worn meantime.

November 26, 1903.—A shade has been worn, over each eye alternately, always for the last five weeks. There is now an alternating convergence of 28°, without glasses.

Advancement right external rectus muscle.

December 4.—Sutures removed. Left external rectus muscle advanced.

December 12.—Sutures removed. Patient kept in bed with both eyes bandaged two days longer.

Since then glasses have been used for near work only. Never any deviation. I have seen the patient frequently, until her marriage and departure for India in March, 1913. She never squints, and, being healthy and fond of outdoor life, the hypermetropia has never troubled her.

CASE A, 503. *June* 7, 1900.—A. girl,[1] aged 5 months, brought to me at the West Ham Hospital. She has squinted constantly with the L. E. since she was twelve weeks old. She has C. S. L. E. about 25°, variable. Fixation lost L. E. Abversion L. E. full. Ordered, continuous occlusion R. E., by a pad and bandage, for fourteen days: also, ung. atropinæ 1 per cent. thrice daily, for both eyes, for retinoscopy.

June 21.—Steady central fixation L. E. Under atropine, angle of squint varies from about 20° to 40°. Retinoscopy R. E. + 5·5 D sph., L. E. + 6·5 D sph.

Ordered, spectacles (of the pattern described on page 103). R. E. + 5 D, L. E. + 6 D. Ordered also, ung. atropinæ 1 per cent., to be put into the R. E. *only* every morning.

August 16.—Glasses worn well, child seems to be quite unconscious of their presence. There is no squint in distant vision. When looking at anything near at hand the infant turns in the R. E. (atropised). Ordered, stop the atropine.

October 8.—No deviation with glasses. When glasses are taken off L. E. turns in to a variable degree —about 30° usually.

May 9, 1901.—No squint with glasses.

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[1] On October 23, 1902, a sister of this patient, an infant aged 14 weeks, was brought to me at the West Ham Hospital. She had C. S. L. E. about 30°. On October 30 retinoscopy under atropine + 6 D sph. each eye. I ordered + 5·5 D and sent her to Mr. Hawes, of Leadenhall Street, who fitted spectacles of the pattern described on page 103. When I saw her on November 6 she had had the spectacles two days. She was wearing them quite happily. Atropine was used for the fixing eye only until the squint alternated. On June 7, 1904, she had no deviation while wearing the glasses, and prism test in dark room showed that the natural development of binocular vision was proceeding.

October 3.—Glasses too small. Ordered, repeat them.

August 28, 1902.—Glasses were lost ten days ago. The child is not squinting now, even without the glasses. Ordered, repeat the glasses.

September 25.—No squint with glasses or without them. When a lighted match was held before the child's eyes in the dark room, she of course looked at it. A prism, apex towards the nose, was then slipped before one eye. This eye was immediately seen to make a slight inward rotation (in order to blend the images of the light), showing that the child had binocular vision.

[I do not think that hypermetropes, during the first three or four months of life, as a rule make any *prolonged* effort of accommodation in the interests of sharp vision. But some infants, this girl, for example, evidently do so. The abnormal accommodative effort caused an abnormal dynamic convergence, which was soon succeeded by a static convergence, before the period at which the fusion sense, normally, has made much progress in development. By giving her a pair of spectacles, and so relieving the strain on the accommodation, the visual axes were, in the course of four or five weeks, brought back to parallelism. This allowed the natural development of the fusion sense to take place. The child, in spite of the high refractive error, is perfectly cured, whether she wears glasses or not. Nothing but an actual muscular paralysis will ever make this child squint again.

The case of this child's sister is exactly similar.

If I had followed the practice recommended in the text-books, of leaving the case until the child was "old enough to wear glasses," she would then have had an incurable squint, and almost total blindness of the left eye.]

Case B, 227. *May* 16, 1900.—A girl, aged 16 years. Her right eye is nearly blind. It turns out

and down, and is very prominent—a hideous deformity —the result of two tenotomies six years ago.

History.—She had whooping-cough soon after she was one year old. During convalescence the R. E. turned in towards the nose. She was taken to an ophthalmic surgeon at once. Being considered to be too young for glasses, she was ordered atropine drops for both eyes. The drops were used for *both* eyes, for from a year and a half to two years. (How often one hears this disastrous tale!) Soon after she was 3 years old she was ordered spectacles. At the age of 10 years the right eye was operated upon, in London (tenotomy of internal rectus). A year later this eye was again operated upon in Germany. After the second operation the R. E. was "straight" for a time. Later it began to turn out and down. This deformity has gradually got worse.

At present R. E. is very prominent: it turns out 28° and down 10°. With this eye she can just distinguish hand movements close to the face. Power of adversion R. E. is absent. She is wearing spectacles + 1·75 D sph. each eye. Vision L. E. $\frac{6}{6}$ both with and without glasses. Retinoscopy—with homatropine L. E. + 2·25 D, R. E. + 3·5 D at approximate macula.

Of course, the blindness of the R.E. is now quite incurable. With a view to remedying the deformity, I advised that the divergence should be dealt with by operation first, and the vertical deviation at a subsequent operation.

May 21.—Having cocainised the eye, I stripped up from the globe the membranes on the nasal side, from near the edge of the cornea to the sunken caruncle. I found the internal rectus muscle behind the sunken caruncle, near the inner wall of the orbit. It was not attached to the globe in any way. As the muscle was much wasted, I decided to advance the conjunctiva, and capsule of Tenon also. I seized all these struc-

tures with forceps, and drew them between the jaws of a Prince's advancement forceps. I secured these structures, by my usual method, to the circumcorneal sclerotic, bringing the eye into a position of very slight convergence.

June 16.—I advanced the right superior rectus muscle.

June 30.—There is still some redness of the eye. The advancements have drawn back the eye into its proper position in the orbit. In ordinary directions of the gaze the eyes appear quite straight. But, when the patient looks more than about 15° to the right, the right eye ceases to follow the movement of the left. This is because, owing to the wasted condition of the internal rectus muscle, I did not dare to rely upon the muscle alone, but was compelled to advance the membranes also. But it is easy for the patient to conceal this defect, by avoiding wide excursions of the eyes. I ordered pince-nez + 2 D sph.

July 5, 1902.—Patient does not wear her glasses out of doors. She always wears them indoors. The eyes are quite natural in appearance.

CASE B, 208. *January* 10, 1900.—A lady, aged 42 years. The right eye is divergent 16°. It turns out more when she is tired. It is more prominent than the left, and the power of adversion is markedly sub-normal. L. E. is hypermetropic to the extent of 1·5 D, and retinoscopy shows about the same defect in the divergent eye. V. L. E.=$\frac{6}{6}$. R. E. has lost fixation and is highly amblyopic. A "dull, dragging ache," which is almost constantly present, troubles the patient even more than the deformity.

History.—When she was a child the R. E. squinted inwards. She wore glasses constantly for several years. At the age of about 10 years the right internal rectus muscle was tenotomised. The eyes were straight for about ten years, when the tenotomised eye

began to turn out. The divergence has increased very slowly ever since. It has been much worse during the last few years.

February 15, 1900.—I advanced the right internal rectus muscle. The muscle was found to be in fairly good condition, and connected to its old insertion by a band of scar tissue.

The patient was kept in bed for eleven days, with both eyes bandaged, the sutures being removed on the ninth day.

May 22, 1900.—The eyes are normally directed and move together normally. There has been no aching in the eyes since the patient left the nursing home.

I have seen the patient at intervals of one or two years. At my request she came to see me again on—

December 16, 1914. (Fourteen years and ten months after the operation.)—The eyes are normal in appearance and free from any discomfort.

Case D, 734. *November* 29, 1901.—Girl, aged 4 years 8 months; attended Moorfields Eye Hospital. C. S. R. E. 22°. Abversion perfect. The power of central fixation of R. E. is lost. The eye is so blind that, when the good eye is tied up, she is unable to see a penny on the floor at her feet. She hears it drop, and goes down on her knees to feel for it. Her mother says that the squint began at about 2 years of age, during an attack of scarlet fever. She says she repeatedly spoke to the doctor about it, but he told her to "wait to see if the child would grow out of the squint"! Ordered, atropine for retinoscopy.

December 3.—C. S. R. E. 26° with atropine. Retinoscopy R. E. + 3 D sph. at approximate macula. L. E. + 3 D sph. Ordered, spectacles + 2·5 D sph. each eye; also L. E. to be continuously occluded by a gauze pad and strapping.

January 3, 1902.—When the eye was at first tied up, she used to fall over things, the mother says.

Later, she could see to run about very well. Child has now central fixation R. E. Ordered, discontinue pad; guttæ atropinæ 1 per cent. to be put into L. E. *only*, every morning. Child to go to school and use the eyes in near vision as much as possible.

February 6.—Child uses (atropised) L. E. in distant vision, and (unatropised) R. E. in near vision. Ordered, continue atropine L. E. *only*.

May 2.—With glasses child uses (atropised) L. E. in distant vision, and (unatropised) R. E. in near vision. When the glasses are off she uses the R. E. and turns in L. E. always. C. S. R. E. 16° with glasses. Ordered, continue atropine L. E.

October 3.—Vision R. E. with glasses $\frac{6}{12}$ easily. C. S. R. E. 13°. Ordered, continue atropine L. E. only, for the first seven days in each month.

December 2.—Child uses (unatropised) R. E. now always, both in near and distant vision, and turns in the (atropised) L. E. C. S. L. E. 14°. Vision R. E. $\frac{6}{9}$ with difficulty; $\frac{6}{9}$ easily; L. E. $\frac{6}{6}$.

April 6, 1903.—C. S. alternating, 15°. No more atropine.

May 3, 1904.—C. S. alternating, 14°. Vision = $\frac{6}{6}$ each eye.

October 6, 1905.—C. S. alternating 8°.

November 7, 1912.—C. S. alternating 10°, with glasses. Vision = $\frac{6}{6}$ each eye.

[It is now certain that the wearing of glasses will not overcome the deviation. The girl and her mother do not consider the disfigurement of any great importance; so it is not proposed to do anything more. It is something to have restored the sight of a nearly blind eye.]

Case A, 541.—[In former editions this was quoted to show how one might deal with such a case in hospital practice, if one had no bed to spare or if the man were unable to leave his work for the necessary length of time. In view of the subsequent history, it

seems an excellent example of what to avoid. Many cases of tenotomy do very well. But, if one had to choose between two evils, I am sure that the average results obtained by leaving all squint cases entirely without operative interference would be better than the average ultimate results of tenotomy.]

August 9, 1900.—A boy, aged 16 years, came to me at West Ham Hospital. He had squinted with L. E. since infancy. He had worn glasses since he was 4 years old. He was wearing + 3·5 D sph. each eye. Vision with glasses, R. E. $\frac{6}{6}$, L. E. $\frac{6}{60}$. He had C. S. L. E. 11°, with his glasses. His glasses were found to be suitable. Adversion L. E. good. Dynamic convergence good. It was, of course, much too late to attempt to restore the sight of the left eye. He wished to have the deformity removed.

Under cocaine and suprarenal extract, I performed complete central tenotomy of left internal rectus by the method described on p. 229.

August 16.—Scarcely any redness remaining. C. S. L. E. 3°, with his glasses. This small squint is quite masked by the angle gamma.

May 8, 1902.—Patient has, with his glasses, C. S. L. E. 4°. There is no noticeable deformity.

February 11, 1909.—In response to a postcard, the patient came to Moorfields Hospital. Eyes are approximately straight.

October 14, 1914.—The patient presented himself at Moorfields after an interval of five and a half years. He says that the left eye began to turn out four or five years ago and had gradually got worse. L. E. is now divergent 14°.

When he can arrange to leave his work for three weeks, I shall have him in the hospital and endeavour to undo what I did fourteen years ago.

Case B, II., 57. *January* 13, 1902.—A girl, aged 11 years 5 months. *History*.—She began to squint

with L. E. when she was 6 years old. She has squinted with this eye constantly ever since. When she was about 8 years old she had glasses. In April, 1899, a surgeon tenotomised the left internal rectus muscle, and in June, 1899, the right also.

She now has convergent squint L. E. 15°, with the glasses. Her vision, with her glasses, is $\frac{6}{6}$ each eye. She has a faint homonymous diplopia when she looks for it. On examining her fusion sense with the amblyoscope she readily blends images, but has almost no amplitude of fusion.

January 24.—Retinoscopy under atropine, each eye, + 3·5 D sph. + 1·5 D cyl. ax. 30° down and out. This very nearly corresponds with the glasses she has been wearing.

February 12.—By my direction the glasses have been left off for the last twenty-four hours. Without glasses, R. E. fixing, the deviation is 22°. L. E. fixing, the deviation is 35°.

I advanced the left external rectus muscle (under cocaine). Eyes "straight" after operation.

February 22.—Removed sutures. Mirror test shows binocular fixation.

February 28.—The patient has binocular vision, both with and without her glasses, but the amplitude of fusion is very small.

June 18.—Since the operation I have, by way of experiment, attempted to increase the amplitude of fusion, but without success. She has now just the same small degree of fusion sense which she had when she began to squint, and which she has had ever since —neither more nor less. But an accurately performed advancement has enabled her to use this feeble fusion sense. The eyes are exactly straight and the child has binocular vision.

The reason of the good vision in the deviating eye is the unusually late onset of the deviation. Amblyopia from disuse is scarcely ever acquired after six years of age.

CASE B, 33. *May* 5, 1896.—Boy, aged 2 years 7 months. The right eye turned in suddenly a little before he was two years old. He has squinted constantly ever since. C. S. R. E. 27°. Central fixation R. E. With R. E. he can with difficulty see the 1½-inch ivory ball at four yards. Abversion each eye perfect. Ordered, atropine for retinoscopy.

May 12.—Under atropine C. S. R. E. 30°. Retinoscopy, each eye + 2·25 D sph. Ordered, spectacles + 2 D sph. constant wear: also guttæ atropinæ 1 per cent. L. E. *only*, every morning.

July 3.—Child uses the R. E. (unatropised) in near vision and the L. E. (atropised) in distant vision. C. S. R. E. 21° with glasses. Ordered, continue.

August 26.—Same. C. S. R. E. 18°. Ordered, continue.

October 12.—The boy uses the (unatropised) R. E., and turns in the (atropised) L. E., nearly always now, even in distant vision. Ordered, continue.

November 6.—C. S. L. E. always now 12°. Ordered, stop atropine.

November 27.—Squint alternates now 9°. Fusion training with amblyoscope.

December 4.—Fusion training. Child readily blends images, but he has at present no amplitude of fusion.

December 10.—Fusion training.

December 14.—Fusion training.

December 16.—Fusion training.

December 19.—Fusion training. Good amplitude of fusion.

January 22, 1897.—No squint.

August 12.—Child wears his glasses. No squint.

October 25, 1898.—Child never squints now.

July 18, 1899.—The boy does not squint even when the glasses are taken off.

June 13, 1902.—Ordered, atropine for retinoscopy.

June 18.—Retinoscopy R. E. + 1·75 D sph. + 0·25

D cyl., L. E. + 1·75 ? sph. Vision $\frac{6}{6}$ each eye. Ordered, glasses for near work + 1·75 D sph. No glasses to be worn out of school. To be seen again in three months.

September 22.—The boy gets on well without glasses. He prefers not to use them even for school work. He never squints now.

CASE B., 165. *July* 19, 1899.—Girl, aged 3 years 5 months. "Neuropathic divergence." Since earliest infancy R. E. had turned out occasionally: worse during the last year. Never any diplopia. Always a delicate and timid child.

The eyes sometimes fix binocularly, but more often R. E. is widely divergent. When the child is spoken to the eyes recover their normal relative directions immediately, but when she looks at a near object the R. E. is usually allowed to diverge. Dynamic convergence very deficient. All separate movements of each eye perfect. With L. E. the child easily sees the $\frac{1}{2}$-inch ivory ball at six yards. With R. E. she can with difficulty see the $1\frac{1}{2}$-inch ball at six yards. R. E. has not lost central fixation. Amblyoscope test shows that the child has some slight degree of fusion sense.

July 26.—Retinoscopy under atropine, each eye + 1·25 D sph. No glasses ordered. Ordered, guttæ atropinæ sulph. 1 per cent. L. E. *only* every morning.

August 22.—Child now uses L. E. (atropised) for distance, and R. E. (unatropised) in near vision. Ordered, continue.

October 27.—Child uses R. E. in near vision and sometimes in distant vision also. With R. E. she can easily see the $\frac{3}{4}$-inch ivory ball at six yards. Ordered, atropine L. E. only every morning, first seven days in each month, for six months.

September 20, 1900.—Divergence is nearly alternating. Still some preference for fixing with L. E.

July 16, 1902.—Divergence almost always present now. V. R. E. $\frac{6}{6}$ partly, V. L. E. $\frac{6}{6}$.

July 29.—Advancement R. internal rectus muscle.

August 26.—Eyes "straight." Child blends images except on looking to extreme R.

[The feeble degree of fusion sense which developed in early childhood was not sufficient to prevent the deviation, or even to cause diplopia, but now that the deviation has been overcome by operation it enables her to fuse the two images.]

I have seen this patient at intervals of two or three years, the last time being on December 3, 1912. There was no deviation. Four-dot test showed that patient had binocular vision.

Case B, 166. *July* 19, 1899.—Girl, aged 8 years 2 months, sister of the preceding case. Alternating divergent squint of variable degree, usually about 35°. Association between accommodation and convergence almost absent. Separate movements each eye normal, except adversion, which is slightly deficient. Fusion sense absent. H.m. 0·5 D. No As. V. each eye $\frac{6}{6}$ Child has worn spectacles + 0·75 D sph. for about three years. Recommended advancement. No glasses required.

August 22.—Advancement R. internal rectus.

November 8.—Advancement L. internal rectus.

December 20.—No noticeable deformity now. Eyes appear to be quite normally directed. Mirror test, however, shows that there is sometimes slight divergence and sometimes slight convergence. Of course there is no fusion : she really uses the eyes alternately, although she appears to use them together.

October 5, 1904.—Condition as in December, 1899.

December 2, 1914. (Fifteen years after the operations.)—Eyes normal in appearance. No discomfort. Vision each eye = $\frac{6}{6}$.

Case A, 79. *December* 5, 1895.—Girl, aged 16

years. Eyes are already under atropine. L. E. is widely divergent. When told to look at a near object she can, by an effort, overcome the deviation, but the eye soon diverges again. Adversion slightly deficient. Patient is known to have been "short-sighted for some years; getting worse." Four or five years ago L. E. turned out, occasionally at first, but during the last year constantly. She often sees double. Small myopic crescent each eye. Choroidal vessels seen. Retinoscopy R. E. −6.5 D sph. V. $\frac{6}{6}$ partly. L. E. 6·5 D sph. −1·75 D cyl. ax. 15° down and out. V. $\frac{6}{9}$. Ordered, full correction to be worn constantly.

February 13, 1896.—When she first began to wear the glasses they made her head ache, but she soon became accustomed to them. Now she finds them quite comfortable. No divergence now. When she is tired she "squints and sees double for a moment until the eyes come straight again." While wearing the glasses either eye diverges when screened, about 20°.

October 8.—Glasses very comfortable. No headaches. No divergence. Behind Maddox rod either eye turns out 12°, varies slightly.

September 23, 1897.—Retinoscopy under atropine shows that the myopia has increased 0·75 D. Ordered, continue same glasses.

July 20, 1899.—She is never seen to squint, but says that she occasionally sees double for a moment. Behind Maddox rod either eye diverges 8° or 9°.

July 27.—Retinoscopy under atropine.—R. E. −7·5 D sph. V. $\frac{6}{6}$ L. E. −7·5 D sph. −1·75 D cyl. ax. 12° down and out. V. $\frac{6}{9}$. Ordered, full correction, to be worn constantly.

August 20, 1903.—Eyes comfortable. Never any divergence. Exophoria is now only 2°.

August 27.—Retinoscopy under atropine shows that the myopia has increased less than 0·5 D during the last four years.

CHAPTER XI

HETEROPHORIA

If a person, with a perfectly normal pair of eyes, looks steadily at any object, both visual axes will continue to be accurately directed to that object, even though one eye be shaded. In other words, his perfectly balanced motor co-ordinations are able to maintain the normal relative directions of the eyes, even when the controlling influence of the fusion sense is temporarily withdrawn. This state of perfect oculo-motor equilibrium is called *orthophoria.*

Heterophoria is the name give to the condition of imperfect oculo-motor balance. There is here a *tendency* for the eyes to deviate from their normal relative directions. Ordinarily, however, this tendency is kept in check by the fusion sense, so that there is no squint. But if binocular vision be temporarily rendered impossible—*e.g.*, by covering one eye—this tendency gives rise to an actual deviation.

Heterophoria of sufficient degree to cause trouble is not very common. Of those who suffer from "asthenopic symptoms," in only a very small proportion of cases are the symptoms found

to be due to heterophoria. Occasionally, however, one meets with a patient who complains of pain and discomfort in the eyes, and whose refraction has repeatedly been examined, and who may have for years worn glasses to correct some unimportant refractive error, without any relief to his suffering. Such a patient usually has a heterophoria, the correction of which immediately and permanently removes his trouble.

Heterophoria may perhaps be due to a muscle or group of muscles being too weak or too strong for the opponents, or to an abnormal position of insertion of a tendon, whereby the muscle acts at less or more than its normal mechanical advantage, or to a muscle or group of muscles being too feebly, or too powerfully, innervated. As a rule, we are unable to determine whether the fault lies in the muscles themselves, or in their innervations. It is very rarely possible to demonstrate any deficiency or excess in the limits of rotation of either eye. We find that the eyes *tend* to assume certain abnormal relative directions, and are seldom able to analyse the defect further than this.

Heterophoria is essentially a defect of motor balance. Squint, on the other hand, is essentially due to a defect of the fusion faculty. In the presence of this fundamental cause, heterophoria may give rise to a permanent squint with suppression of one image : not otherwise.

There is an apparent exception to this rule—a person who has previously enjoyed perfect binocular vision may have the visual acuity of one eye so lowered by progressive myopia, injury, or disease as to render binocular vision impossible. Any heterophoria which may be present will then cause a manifest squint, although the cerebral faculty of fusion remains perfect.

Heterophoria is the generic name, invented by Stevens, for all latent tendencies to deviation. Distinctive names are employed to indicate the direction of the tendency :—

Esophoria is a tendency to abnormal static convergence of the visual axes.

Exophoria is a tendency to divergence of the visual axes.

Hyperphoria is a tendency of the two eyes to rotate vertically in opposite directions, so that one visual axis shall lie in a higher plane than the other. The eye which tends to turn upwards relatively to the other is called the hyperphoric eye.

Cyclophoria is a tendency to abnormal rotation of the eyes round a fore-and-aft axis, so that what should be the vertical meridian of the eye shall be no longer parallel to the median plane of the head. A tendency for the vertical meridians of the eyes to lean away from the median plane is called plus cyclophoria. A tendency in the opposite direction is called minus cyclophoria.

Pseudo-heterophoria and latent heterophoria.—In a case of uncorrected ametropia there is frequently an apparent heterophoria which disappears when the appropriate correcting glasses have been worn for some time. The term heterophoria should be reserved for cases in which the anomaly persists after optical correction of any refractive error which may be present.

If the patient be ametropic, he should wear an exact correction of his ametropia during the examination. But this spurious heterophoria does not always disappear immediately on correcting the refractive error. So that, if the latter be considerable, one should not immediately conclude that any heterophoria which may be found is genuine. The results should be checked by a second examination after glasses correcting the refractive error have been worn for several weeks. Similarly, after glasses correcting a refractive error have been worn for some weeks, one sometimes finds an esophoria or exophoria which was not manifest before.

When for any reason an eye has been occluded for several weeks one may usually find a considerable degree of oculo-motor imbalance. This may be a heterophoria which was previously latent; but I think not, because it gradually disappears and there are then no symptoms suggesting heterophoria.

The *symptoms* of heterophoria are those of "eye-strain" in general—frontal headache coming on towards the end of the day; pain in the eyes

after watching anything intently, *e.g.*, a play; migraine; dizziness (especially associated with hyperphoria); conjunctival hyperæmia, etc. In the higher degrees of heterophoria momentary deviation with diplopia is not uncommon.

Asthenopic symptoms, which do not yield to accurate optical correction of any refractive error which may be present, should always lead to investigation of the motor balance of the eyes, if this has not already taken place.

People vary greatly in their susceptibility to suffering as the result of heterophoria, just as they do in the case of refractive error. Other things being equal, hyperphoria is the form of heterophoria which is most likely to cause trouble, and esophoria the least. It is not uncommon to see a patient who has several degrees of esophoria, and who is quite unconscious of any defect; whereas few people can support a hyperphoria of more than 1° without inconvenience.

The importance of a case of heterophoria is proportionate to the trouble which it causes. A case which gives rise to no symptoms requires no treatment.

Heterotropia.—A person whose fusion sense has developed perfectly, but who has a very high degree of heterophoria, will be able (with more or less suffering) to keep this deviation-tendency in check during the adaptable and vigorous period of childhood and youth, but, when he exchanges

school life for some more trying and less healthy occupation, he may find himself unable to continue the struggle, in which event his heterophoria gives rise to an actual deviation. He then loses his asthenopic symptoms, but he suffers from diplopia, which is usually so annoying that he is glad to shade or close one eye. The degree of the manifest deviation increases during the first few weeks or months, after which it becomes stationary. The term heterotropia should be reserved for this rather rare condition, as it is obviously a further stage of heterophoria, and not a true squint nor a paralysis. The case of Mr. S. H., p. 199, is a typical example.

The methods of testing the relative motor balance of the eyes.—In a case of heterophoria, under ordinary circumstances, the desire for binocular vision prevents the eyes from deviating from their normal relative directions. But if, by artificial means, the image formed in one eye be so altered in appearance or position as to make fusion with the other unaltered image impossible, the control of the fusion sense is suspended. The heterophoria then gives rise to a manifest deviation. The altered image in the deviating eye is not suppressed as in a case of squint. The diplopia, therefore, gives an easy means of ascertaining the direction and degree of the deviation. This is the principle on which all subjective tests for heterophoria are based.

The *instruments* required for the tests which I am about to describe are the Maddox rod and double prism, an adjustable trial frame, test cards, and a set of prisms whose axes are accurately marked. An *accurate* rotary prism also is very useful.

The Maddox rod (fig. 20).—A transparent round glass rod is, in effect, a very strong cylindrical lens. Rays of light, therefore, which pass through it are dispersed in one plane only, at right angles to the

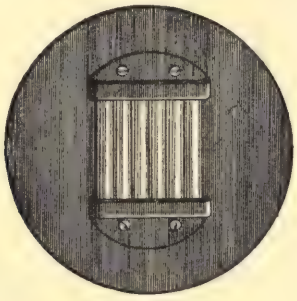

Fig. 20.

axis of the rod. So that if a point of light be looked at through this rod, it will appear as a long, narrow band of light. For the sake of convenience, half a dozen of these pieces of glass rod are fixed, side by side, in a metal disc of such a size as to fit into an ordinary trial frame. The rods may be made of red glass, to increase the contrast between this band of light and the true image. An equally good plan is to have the rods made of colourless glass and to put a plane red glass before

the other eye. This test, owing to its simplicity and general reliability, is quite the best for ordinary clinical use. I have, however, found some cases of heterophoria in which there appeared to be a tendency to blend a part of the streak with the point of light.

The source of *light* should, if possible, be a frosted incandescent electric lamp, enclosed in a box having a small round opening facing the observer. Failing this, any bright flame, enclosed in a tin chimney having a hole of about $\frac{3}{4}$ inch diameter in the side of it, will serve the purpose. The general illumination of the room should be very subdued.

As the rods produce extreme distortion in one direction only, a well-marked vertical line can be seen through the rods when their axes are horizontal, and a horizontal line can be seen when their axes are vertical. Care should therefore be taken that there be no long horizontal or vertical lines near the centre of the field of vision.

In looking at the light, with the rod before one eye and the other eye naked, the naked eye will of course see the light and the surrounding objects; but, to the rod-clad eye, the light will appear as a long streak, and less luminous objects will not be visible at all. It is not possible to blend two such dissimilar images as the round point of light and the streak, so the function of fusion is temporarily suspended and the eyes are merely

controlled by their motor co-ordinations. If there be no motor anomaly the streak, seen by the rod-clad eye, will appear to pass through the flame, seen by the naked eye. But if there be any heterophoria it will now be able to cause the eyes to deviate, the relative positions of the streak and light indicating the direction and degree of the deviation.

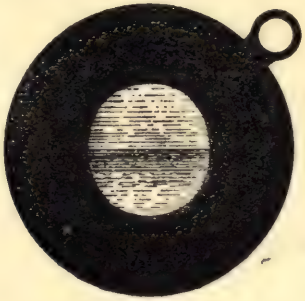

FIG. 21.

The Maddox *double prism* (fig. 21) consists of two prisms, each of 4°,[1] ground base to base on the same piece of glass. When this double

[1] To avoid confusion, the *strength of a prism* is always, in this book, denoted by the *number of degrees which it deflects a ray of light.* Chromatic dispersion produces no appreciable error in the weak prisms used in ophthalmology.

An optician usually numbers a prism according to the width of its geometrical angle (the angle between the two plane surfaces). The refracting power of such a prism varies according to the kind of glass of which it is made. For practical purposes it may be taken as half the geometrical angle. For instance, a prism which deflects a ray of light to the extent of 4° will have a geometrical angle of about 8°.

prism is placed with its apices vertical, before one eye, so that the line of junction of the bases crosses the pupil horizontally, two false images of any small object will be seen, one above and the other below its true position. If now the other (naked) eye be opened, it will see the real image of the object mid-way between the two false images. The false images are not changed in appearance, but the vertical displacement of each is too great to admit of the true image being blended with either of them. The eyes are thus temporarily deprived of the control of the fusion sense and abandoned to their motor co-ordinations, just as in the last test.

In looking at a horizontal line, the true image can of course not be made to approach either of the two false images ; but there must be no long vertical line near the centre of the field of vision.

The *test cards* which I use consist of two pieces of strong white cardboard each 2 feet square. Number I. card has in its centre a straight black line 2 inches long. In the centre of Number II. card are ten letters of "pearl" type having a large capital O in the middle. The cards are meant for use at the reading distance. The reason for making them so large is that the objects shall be seen in the centre of a blank field with no edges near to solicit fusion.

In the examination I begin with the Maddox rod. The patient is seated facing the box which

Number I. Test Card.

FIG. 22.

Number II. Test Card.

FIG. 23.

contains the test light, at a distance of at least five or six metres from it. If he be not absolutely emmetropic, he wears correcting lenses in the trial frame in every test. The frame is adjusted so that the lenses are accurately centred for distant vision. The rod, with its axis horizontal, is put in the frame before the right eye. The test light is switched on. If the vertical streak, seen by the right eye, appears to go through the light, seen by the left eye, the patient has no esophoria or exophoria in distant vision. Now rotate the rod so that its axis is vertical. If the horizontal streak, now seen by the right eye, appears to pass through the light seen by the left eye, the patient has no hyperphoria in distant vision.

Now remove the rod and replace it with the double prism, with apices vertical (line of junction of bases horizontal). Adjust the trial frame for near vision. Let the patient hold in his hand Number I. test card, with the line horizontal. He will see two false images of the horizontal line with the prism-clad eye, and between them he will see the true image with the naked eye. If the middle line appears equidistant from each of the other lines, and has its ends level with their ends (fig. 24), there is no hyperphoria, esophoria, or exophoria in near vision. If the middle line appears parallel to the other two lines, as in this figure, there is no cyclophoria. The patient's

oculo-motor equilibrium, therefore, is perfect in every respect.

It should be noted that, with orthophoria in distant vision, a little exophoria—perhaps 2° or 3°—appears to be the normal average condition in near vision.

If any anomaly be found during these proceedings, further examinations will be required.

In the *distant vision test* with the Maddox rod, axis horizontal, before the right eye, if the vertical streak lies to the right of the light (homonymous

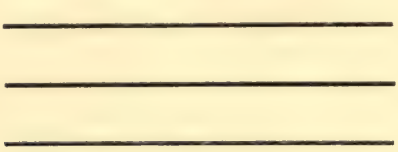

FIG. 24.[1]

diplopia), there is esophoria. If it lies to the left of the light (crossed diplopia), there is exophoria. The degree of the defect is measured by placing before the left eye a prism, base out in esophoria, base in in exophoria, of such a strength as to cause the streak to pass through the

[1] This and the two succeeding figures are taken from a paper on "Insufficiency of the Obliques," by Dr. Savage, of Nashville, U.S.A., *Archives of Ophthalmology*, January, 1891. "Ophthalmic Myology," by the same author, contains the fullest account of cyclophoria which has hitherto been published.

light. As a control test, remove the prism and change the rod from the right eye to the left. The position of the streak also changes over, that is, it still shows the same kind of diplopia. The degree of heterophoria indicated is the same as before. Now place before the right eye the prism which corrected the defect when placed before the left eye. This should again cause the streak to pass through the light.

In using the rod, axis vertical, before the right eye, if the horizontal streak is seen below the light, this indicates that the right eye tends to turn upwards relatively to the left eye (right hyperphoria). If the streak is seen above the light, the left eye tends to turn upwards relatively to the right eye (left hyperphoria). Place before the left eye a prism, base up in right hyperphoria or base down in left hyperphoria. The strength of the prism required to bring the streak through the light measures the degree of the hyperphoria. Now remove the prism and change the rod from the right to the left eye. Almost invariably, if the right eye saw the streak above the light, the left eye will now see it below, and *vice versa*. That is to say, the hyperphoria is comitant. The measurement should now be checked by neutralising the hyperphoria with a prism placed before the right eye.

In rare instances each eye, in turn, rotates upwards behind the rod (anaphoria).

In the *near vision test* with the double prism before the right eye, and the Number I. test card, the middle line, seen by the left eye, should be equidistant between the two false images, seen by the right eye. If it lies nearer the upper false image, there is right hyperphoria. If it lies nearer the lower false image, there is left hyperphoria. The prism, base down, before the hyperphoric eye, which places the line half-way between the two false images, will serve to measure the degree of the hyperphoria in near vision.

If the three lines are not all level with each other at the ends, hand the patient Number II. test card and tell him to read the letters on it. The small letters are to ensure a normal effort of accommodation. If presbyopic, the patient is allowed glasses. He will see the true image of the object between its two false images. In orthophoria the three images will be in the same vertical line. If the middle image, seen by the left eye, is to the left of the two false images, the patient has esophoria in near vision. If it is to the right, he has exophoria in near vision. The prism, axis horizontal, which will bring the three O's in line, is a measure of the defect. In near vision it is better to measure with prisms than to use any kind of small tangent scale, because with the former the distance from the eye is of no consequence, whereas with the latter the smallest variation introduces an error. The study of esophoria and exophoria in near vision is

intimately associated with that of convergence anomalies.

When the patient looks at the horizontal line on Number I. card with both eyes open and the double prism before the right eye, the three lines should be parallel (fig. 24). If not, there is cyclophoria. Suppose the middle line, seen by the left eye, seems to dip down to the left as in fig. 25: this shows that the vertical meridians of the eyes

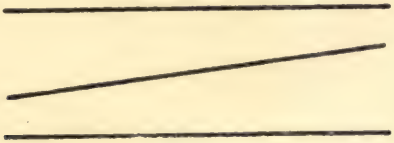

FIG. 25.

FIG. 26.

are leaning in the opposite direction, towards each other (minus cyclophoria). If the middle line, seen by the left eye, appears to dip down to the right as in fig. 26, there is plus cyclophoria.

Prism duction should always be investigated in any case in which heterophoria has been found.

Seat the patient at a distance of five or six metres from a candle flame. Let him wear a trial frame. While he looks steadily at the light, put a

1° prism, base down, before the right eye. Gradually increase the strength of the prism, until the highest prism is found which the patient can bear without seeing double. This indicates the extreme range of superduction of the right eye. Now test the superduction of the left eye. The subduction of each eye is similarly tested, with prisms base up. The power of binocular abduction is tested with prisms, bases in. Binocular adduction is so intimately associated with accommodation that an attempt to measure it with prisms (which cause the eyes to converge without accommodating) gives very variable and misleading results.

The normal limits of prism duction are as follows :—

Superduction ...	...	$1\frac{1}{2}°$ to $2\frac{1}{2}°$
Subduction ...	...	$1\frac{1}{2}°$ to $2\frac{1}{2}°$
Abduction ...	...	4° to 5°

No amount of practice appears to increase the duction power in these three directions. Convergence, on the other hand, can nearly always be much increased by practice. As the degree of prism duction does not vary from time to time, and is independent of voluntary effort on the part of the patient, the information obtained is reliable.

A rotary prism (fig. 27) is very convenient for measuring duction. It consists of two prisms of equal strength, mounted in a metal disc in such a

position that the apex of each coincides with the base of the other. In this position they, of course, neutralise each other. By a mechanical arrangement, the two prisms can be rotated in opposite directions at equal rates. The strength of the compound prism can be thus gradually increased from zero up to the combined strength of the two components.

FIG. 27.

Phorometer.[1]—Fig. 28 shows an instrument which I devised for measuring heterophoria. It is very reliable, and easy to use. A rod-shaped wooden box 24 inches by 2 inches by 2 inches is supported on a stand by a horizontal bolt, so that it is free to rotate in a vertical plane. (A two-foot length of brass optical tube would do as well as the rod-shaped box.) In the face of this rod-shaped box are three openings, each 3 inches by $\frac{5}{8}$ inch. In the centre opening is fitted a piece of frosted red glass. In each of the two

[1] Made by Mr. Hawes, 79, Leadenhall Street, London, E.C., and by most manufacturing opticians. I have improved it by substituting for the front of the rod-shaped box a round target of American "3-ply," which is painted black. The three lights are thus shown on a black field.

other openings is a piece of frosted green glass. In the box, behind each glass, is a small electric lamp having a tin reflector behind it to increase the illumination. The box is ventilated by holes in the back. On the back of the stand is a brass protractor, marked in degrees, to show the axis at which the rod-shaped box is placed.

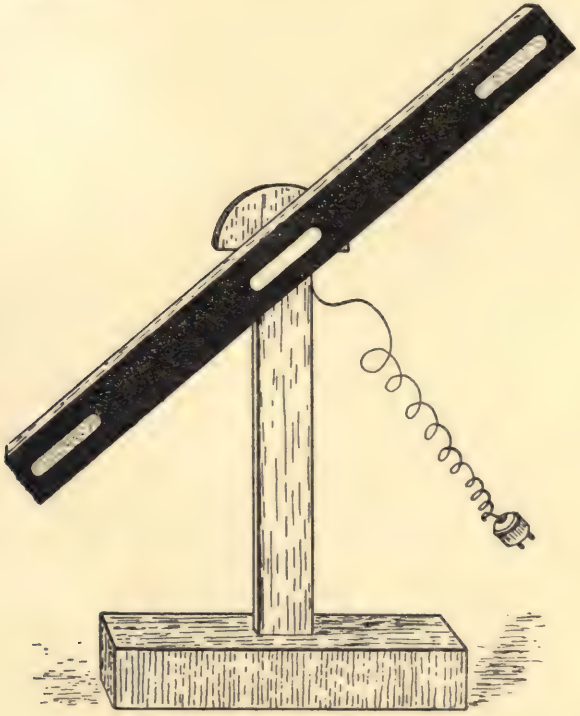

FIG. 28.

The room is partially darkened by drawing down the blinds. The patient is seated in front of the instrument at a distance of about 8 or 10 feet. In a trial frame he wears a red glass before the right eye, and a green glass before the left. With the right eye he sees nothing but the centre red light; and with the

left eye he sees nothing but the two green lights. The angular distance between the lights is sufficiently *great to avoid any tendency to fuse the red light with either of the green lights,* so that the eyes are perfectly dissociated. First the rod-shaped box is put horizontal and the patient is asked whether the three lights appear in the same straight line. If they do, he has no hyperphoria. If the red appears below the level of the green lights, he has right hyperphoria. If it appears above, he has left hyperphoria. The degree of the defect is measured by putting a rotary prism (fig. 27) in the trial frame and screwing it up until the lights appear in line. Esophoria and exophoria are similarly detected and measured by placing the rod-shaped box vertical—red to the right indicating esophoria, and red to the left, exophoria.

If one wishes to ascertain the direction of a compound defect, such, for instance, as esophoria with right hyperphoria, the box is rotated until the lights are in the same straight line, though of course not equidistant from each other. The axis is then read off from the protractor at the back of the stand. To ascertain the degree of the compound defect, place the box with its axis at right angles to this, then screw up the rotary prism until the lights are again in the same straight line.

Esophoria.

Low degrees of esophoria are very common and are usually of no importance. The liability of an esophoria to cause trouble is determined not so much by its degree as by the condition of the functions of abversion and binocular abduction. A patient who can abvert each eye separately until the cornea touches the outer canthus, and whose binocular abduction is not less than 3°, will as

a rule be able to support many degrees of esophoria without inconvenience.

In many cases of esophoria of high degree there is occasional momentary diplopia. When the patient is gazing vacantly without perceiving what is before his eyes, perhaps the visual axes may deviate. Instantly the diplopia awakens the dormant fusion sense, and the eyes immediately recover themselves. These cases are often mistaken for occasional convergent squint. Occasional squints, however, differ from esophoria in that the fusion sense is defective, so that the deviation is not so instantly corrected, and diplopia is either absent or very faint. In an occasional squint, too, examination with the Maddox rod shows that when the deviation is not actually present there is little or no tendency to convergence. In a case of occasional squint we have a pair of eyes, not properly controlled by the fusion sense, responding to intermittent and varying nervous impulses. In esophoria there is a constant and definite motor anomaly which is kept in check by a perfect fusion faculty.

Treatment.—Moderate degrees of esophoria do not cause inconvenience unless the binocular abduction is very deficient. In these cases the symptoms are relieved by the constant wearing of prisms, bases out, which represent the deficiency of binocular abduction (*not* the degree of esophoria). The prism should be divided between

the two eyes. For example, a patient with 5° of esophoria may not be inconvenienced thereby. But if his binocular abduction is only 2° instead of 4° he is likely to suffer from "asthenopic" symptoms and occasional diplopia. He will be relieved by wearing prisms, bases out, having a total deviating power of 2°. If the patient already wears glasses for correction of a refractive error, the prismatic effect may perhaps be got by decentration of lenses (see Appendix).

In a case of esophoria of high degree, if treatment be required, advancement of an external rectus muscle should be performed.

Exophoria.

Uncomplicated exophoria of moderate degree seldom causes any inconvenience. But if, as occasionally happens, there is a defect of dynamic convergence in addition, the patient is likely to suffer from frontal headache, not only after using the eyes in near vision, but often at other times also. Patients who have exophoria of high degree frequently suffer from pain in the eyes during reading, and the eyes may momentarily deviate, but this is less common than in esophoria.

Treatment.—Prisms are seldom of use in exophoria. Slight cases require no treatment, if uncomplicated. Prisms of sufficient strength to correct high degrees are seldom tolerated. If the

case be complicated by convergence deficiency, treatment should be directed to this anomaly. Even if there be no deficiency of dynamic convergence, convergence training often relieves the patient's symptoms, but, in my experience, the effect has been only transitory. In higher degrees of exophoria, especially if adversion be deficient, one internal rectus muscle, or both, should be advanced. One should then aim at producing an operative effect exactly equal to the degree of the exophoria in distant vision. Exophoria is almost invariably due to underaction of the internal recti, scarcely ever to overaction of the externi. For this and other reasons, tenotomy of a rectus externus is not advisable.

Hyperphoria.

Clinically, hyperphoria is the most important of all forms of heterophoria, because of the severity of the symptoms to which it is liable to give rise, and the certainty with which these symptoms may be relieved. The liability of a case to cause trouble depends not only upon the degree of the hyperphoria ; but upon the extent of any deficiency of prism duction in the opposite direction. For example, an ordinary healthy man who has 1° of right hyperphoria will probably suffer no inconvenience if the right subduction is as much as 2° ; but if it is only 1° or less he is almost certain to have trouble.

The commonest symptoms are aching in the

eyes and frontal headache, coming on towards the end of the day, not especially caused by near work. Some patients complain of giddiness on looking down. Momentary diplopia is not uncommon.

In a marked case of hyperphoria, more often than not, the palpebral fissure on the hyperphoric side is smaller than that on the other. This asymmetry disappears entirely when the hyperphoria is corrected.

One is not infrequently asked, "How do you know which eye is at fault?" Neither eye is at fault. Hyperphoria is not a defect in the motility or balance in either eye. It is a defect in the *relative* motor balance of the two eyes. As an illustration, put on a spectacle frame with a 2° prism, say, base up before the right eye. One now has an artificial left hyperphoria of 2°. One immediately experiences a strained, aching sensation in the eyes, which becomes more and more unpleasant until, after a time, it causes a rather severe headache. But no defect in motility can be discovered in either eye, and the prism may be put the other way up or transferred to the other eye without producing any alteration in the symptoms or in the limits of rotation of either eye.

The cause of the pain is not quite clear. It is obviously not due to any strain upon the muscles themselves. It must be due, in some way, to the effort required to innervate unequally two conjugate sets of muscles.

The *treatment,* in any case of hyperphoria of moderate degree, is by prisms to be worn constantly. The prism should be placed, base down,

before the hyperphoric eye. Or, if more than 1° is required, the effect may be divided between the two eyes, the prism before the other eye being placed, of course, base up. The strength of the prisms should be determined partly by the degree of the hyperphoria and partly by the range of prism duction (see p. 183). One should attempt as nearly as possible to correct the hyperphoria and to bring each eye into the middle of the vertical range of prism duction. For example, take a case of right hyperphoria 2°, in which the right superduction (and left subduction) is 3° and the right subduction (and left superduction) is 1°. A prism, base down, before the right eye, 2° would correct the hyperphoria; but a prism of 1° would suffice to bring the eye into the middle of the range of prism duction. In this case one would order a prism of 1½° as a compromise.

If the hyperphoria is very high—over 4°, for example—the correcting prisms would be inconveniently heavy; therefore operation is usually required. If a hyperphoria of lower degree causes the patient sufficient discomfort to make him desire treatment, and if he especially wishes to avoid the necessity of wearing glasses constantly, the case may be cured by operation. If there be any doubt whatever about the hyperphoria being the cause of the symptoms, the patient should wear the correcting prisms for a few days in order to

decide this point. Some of the hyperphoria may be latent. Therefore one or other eye should be occluded by a patch for a week before the operation. Usually the manifest hyperphoria increases slightly during the first four or five days, and then remains stationary. Then the inferior rectus muscle of the hyperphoric eye should be advanced. If accurately performed, this is certain and safe. Tenotomy of a superior rectus is not safe. Operation in a case of hyperphoria is not to be undertaken lightly, or by a surgeon of small experience, for any inaccuracy may lead to disaster.

Anaphoria.

This name is applied to the rather rare condition in which either eye turns up when screened. It has been called "double hyperphoria," but the term is open to objection. I have notes of a few well-marked cases. In all there was more or less drooping of both eyelids. A distinct effort was required to open the eyes widely while looking straight ahead, but in looking up, the lids were lifted normally. Subduction of either eye was normal. These cases were possibly due to a faulty nervous connection between the superior recti muscles and the levatores palpebræ superiores.

I have seen several cases of anaphoria associated with plus cyclophoria—due probably to underaction of the superior oblique muscles.

Anaphoria coexisting with convergent squint may

easily be distinguished from apparent vertical deviation (see p. 35).

I have no note of any case of kataphoria (downward tendency of each eye).

Cyclophoria.

In order that binocular vision may be possible it is necessary not only that the visual axes of the two eyes shall be directed to the same object, but that their vertical diameters shall be parallel. The work of keeping the vertical diameters parallel falls mainly upon the oblique muscles. If the superior obliques act too feebly, the eyes tend to rotate round a fore-and-aft axis so that their vertical diameters diverge above (plus cyclophoria). This is very much more common than minus cyclophoria. Plus cyclophoria is not infrequently associated with anaphoria.

Cyclophoria may cause giddiness, and difficulty in judging the true position of the steps in going downstairs.

Not much can be done in the way of direct treatment in cases of cyclophoria. But there are some practical points which deserve careful study.

Close one eye and hold before the other a strong convex cylindrical lens, axis vertical. Look at a horizontal line, the junction of a floor and wall, for instance. The horizontal line still appears horizontal. Now rotate the lens a few degrees.

The horizontal line appears to rotate slightly with the lens. In other words, the cylindrical lens rotates the image of the line towards its meridian of greatest convexity.

Next put on a trial frame and place before each eye a + 1 D cylindrical lens, axis vertical. Vision will be slightly blurred, but objects will not appear displaced, and the lenses can be worn for a long time without discomfort. Now rotate each lens about 30°, so that their axes diverge above. On looking down, the floor seems far away, and, on looking up, the ceiling seems quite near—one feels about 7 feet high. Now rotate the cylinders in the opposite direction, so that their axes converge above. The floor appears quite near, and the ceiling high—one feels a dwarf. After a few minutes of this artificial oblique astigmatism one experiences a feeling of giddiness and nausea.

In the light of the former experiment, the explanation is plain. We are accustomed to localise all objects with reference to the horizontal surface (floor, ground, or sea) which supports us. While wearing the convex cylinders with axes divergent above, the images of the horizontal surface of the floor are tilted outwards towards each temple. In order that these images may be received upon corresponding points of the two retinæ, each eye must rotate about a fore-and-aft axis so that the vertical diameters diverge above. This is accomplished mainly by a lessened action

of the superior oblique muscles and an increased action of the inferior obliques. But this has also the effect of rotating the two eyes a little upwards, so that, in looking down to the floor, one has to put forth sufficient energy to overcome this upward tendency, as well as to effect the actual downward rotation. And in looking at the ceiling less than the normal expenditure of energy is required, owing to the eyes already having an upward tendency. We depend chiefly upon the "muscular sense" of the external ocular muscles in judging the relative positions of objects. Therefore, the increased effort required in looking down makes the floor appear farther away, and the lessened effort in looking up makes the ceiling appear lower.

In the experiment with the axes of the cylinders convergent above, the conditions are exactly reversed.

This experiment appears to explain the commonly observed fact that astigmatism is more liable to cause trouble when the axes are oblique than when they are vertical or horizontal. Uncorrected astigmatism, unless the axes in the two eyes are parallel or at right angles, must cause a pseudo-cyclophoria, which should disappear when the astigmatism is corrected.

Now and then one meets with a patient with oblique astigmatism, who is less comfortable with glasses which accurately correct his refractive

error than he was without any correction at all. In such a case, one generally finds that he has cyclophoria of an opposite kind to the pseudo-cyclophoria which would be produced by his uncorrected astigmatism. So that they, to a certain extent, neutralised each other. But, with the correction of his astigmatism, the whole of his true cyclophoria becomes manifest. A slight rotation, say about 5°, of both his cylinders, in the direction which favours the feebly acting pair of oblique muscles, will often make him quite comfortable without appreciably lowering his visual acuity.

Under certain conditions it is possible, without any rotation of the eyes round a fore-and-aft axis, to blend images of lines which are slightly tilted in opposite directions (see pp. 11 and 12). Some authors have therefore assumed that the eyes never make an axial rotation in the interests of binocular vision. This view does not accord with clinical and experimental evidence. Fusion of tilted images takes place according to the law stated on p. 11.

Rhythmic exercises with prisms, cylinders, etc., are employed by many eminent ophthalmologists in the United States of America, so it seems certain that they must do good in some cases. But I have tried all the most approved methods, and have never been able to satisfy myself that I have produced any effect in any case of esophoria or hyperphoria. Cases of exophoria are indirectly benefited by exercising the dynamic convergence, but that is a different matter.

In America there may be a tendency to over-estimate the importance of small latent deviation tendencies. But this is less harmful than the almost total neglect which the subject meets with in this country.

Here are some examples :—

Mr. R. H., aged 38, a hard worker, and head of a large city business, consulted me on October 17, 1899. He complained of dull, aching pains in the eyes and forehead. Pain was always relieved by sleep. It was not especially associated with near work. He suffered almost as much during his holidays. His eyes had been examined many times, and he had worn glasses for twelve years. There was a slight drooping of the left upper lid. He was wearing + 2·25 D sph. each eye. He showed me several prescriptions for glasses, all practically the same. Retinoscopy without mydriatic showed 2 D of hypermetropia each eye, no astigmatism. His corrected vision was $\frac{6}{6}$ easily, each eye. On investigating the motor balance of his eyes, I found he had nearly 2° left hyperphoria. Left superduction and right subduction were each 4°; and left subduction and right superduction were each 1°. I ordered spectacles + 2 D sph. each eye, the right lens to be combined with a $\frac{3}{4}$° prism, base up, and the left lens with a similar prism, base down.

On September 26, 1902, I saw the patient again. He has worn the glasses constantly, with perfect comfort, and is entirely free from the old trouble.

On September 11, 1912, the hyperphoria was of exactly the same degree: patient has worn the same prisms throughout, and is quite free from discomfort.

Miss E. B., aged 33, seen with Mr. Devereux Marshall on February 6, 1903. Patient had suffered from

frontal headache and occasional diplopia for as long as she could remember. Headaches were always relieved by sleep, were independent of occupation, and became more severe towards the end of the day. She had for many years worn an exact correction of her ametropia (+0·75 D cyl. ax. vert. each eye), but the glasses gave her no relief.

On investigating the motor balance of the eyes we found left hyperphoria 4°, exophoria 1°. Right superduction and left subduction barely 1°. Right subduction and left superduction each 6°. We ordered, for constant wear, to be incorporated with her cylinders, R. E. prisms 1½°, base up, L. E. prism 1½°, base down.

The patient has been seen twice since that date. She is perfectly comfortable and entirely free from headaches.

Miss F. L., aged 17, consulted me on February 2, 1900. She was wearing spectacles + 1 D sph. She said that when she was tired her eyes would often be crossed for a moment and she then would see double. The crossing of the eyes only lasted an instant. She had frequent frontal headaches. She had worn glasses since she was ten years of age. She had been told that she suffered from "periodic strabismus." Retinoscopy without mydriatic showed hypermetropia of 0·5 D only. Vision of each eye $\frac{6}{6}$. Examination with the Maddox rod showed esophoria, 9°, in distant vision. Esophoria, in near vision, was 8°. Binocular prism abduction was only 2°. R. E. could be abverted until the cornea touched the outer canthus. L. E. abversion not quite so complete. The fusion faculty was perfect. The case was not one of "periodic strabismus" at all, but an example of esophoria of high degree.

February 5, 1900.—Retinoscopy was confirmed under atropine.

February 20, 1900.—I advanced the left external rectus muscle.

February 27, 1900.—Stitches removed.

March 29, 1900.—Patient has 1° esophoria in distant vision, perfect orthophoria in near vision, binocular prism abduction 5°. Patient never has diplopia now.

June 18, 1902.—Patient has not been seen to cross her eyes since the operation, and she has had no diplopia. She is quite free from headaches now. She has, of course, not worn glasses since the operation.

Mr. S. H.—In 1905 a gentleman, aged 20, was sent me by Dr. Bolton Tomson. From early childhood until one year before he came to me the patient had suffered from severe headaches coming on towards the end of the day. He had had occasional diplopia, one image being over the other. He had also suffered from frequent attacks of typical migraine. During the last year he had seen double constantly, and the right eye had squinted downwards. He could still, by a great effort, overcome the deviation and blend the images. The diplopia was so intense that he was only comfortable when one or other eye was covered. But *since the hyperphoria had given place to an actual deviation, the headaches and migraine had completely disappeared.*

There was no important refractive error. R. E. deviated downwards (or L. E. upwards) 8°. Either eye deviated outwards 5°. Prisms of this strength gave binocular vision with orthophoria.

Treatment consisted in advancement of the L. inferior rectus muscle. If this operation had been performed many years earlier the patient would have been spared much unnecessary suffering.

Insufficiency of Dynamic Convergence.

This is not a heterophoria, but it is convenient to discuss it in this chapter.

There has been much confusion on the subject of insufficiency of convergence, because authors have not clearly distinguished between static and dynamic convergence.

A person whose visual apparatus is normal has no static convergence at any time; in distant vision he exercises no dynamic convergence; in near vision his dynamic convergence exactly suffices to cause both visual axes to be directed to the near object. If his dynamic convergence were excessive, there would be a *tendency* to convergent squint in near vision; if it were markedly insufficient, he would have difficulty in maintaining convergence, as in reading, for instance, for any length of time.

A person who has a convergent squint which persists after correction of any refractive error has an unchecked static convergence; one who has esophoria has a static convergence which is kept in check by constant muscular effort,[1] evoked by the desire for binocular vision. Static convergence is a minus quantity in divergent squint. In exophoria there is a minus static convergence which is neutralised by an abnormal effort of

[1] This "without prejudice" to the much debated question of the existence of a cerebral centre for divergence.

dynamic convergence. In these cases the power of dynamic convergence may or may not be normal.

A patient, therefore, who has exophoria in distant vision and the same degree of exophoria, or less, in near vision, cannot properly be said to have insufficiency of convergence. This distinction between exophoria and insufficiency of dynamic convergence is of great practical importance, because the treatment of the two affections differs entirely. In the former case, if any treatment be required at all, operation is usually necessary ; in the latter, operation is always contra-indicated, and benefit may often be obtained from exercises.

Insufficiency of dynamic convergence, apart from neuropathic cases, is not common.

The symptoms produced by insufficiency of convergence are pain in the brow after reading, and a tendency to hold the book at a long distance from the eyes (apart from any error of static or dynamic refraction).

Convergence being a voluntary act, the extreme degree of which any individual is capable will vary from time to time, according to the state of his health and the amount of energy he is able to put forth at the moment. Elaborate instruments for determining the near point of convergence are, therefore, not required. Moreover, this information is not of much practical use.

The best procedure is to test the horizontal motor balance of the eyes, first in distant vision, and then at ten inches. If there is no more exophoria, or no less esophoria, in near vision than there is in distant vision, the patient has no insufficiency of convergence. If the patient has orthophoria in distant vision and exophoria in near vision, or if there is more exophoria, or less esophoria, in near than in distant vision, he theoretically has insufficiency of dynamic convergence of a degree equal to the difference. But a difference of 2° to 3° is found in the majority of people; so an "insufficiency" which does not exceed 3° should not be regarded as abnormal.

In an uncomplicated case of insufficiency of convergence, exercises should be tried. The following procedure is as good as any: Any error of static or dynamic refraction is corrected by glasses. The patient begins reading a book at the ordinary distance. Then, while still reading, he gradually brings the book nearer his eyes until the print begins to be blurred. He then slowly removes the book to the ordinary reading distance. This is repeated. At about every tenth line he looks into the distance for a moment, in order completely to relax his convergence. Two or three pages should be read in this way, three or four times a day for a month. This simple plan has given quite as good results as the

more elaborate methods which I have tried. This is perhaps because the patient finds it more convenient to carry out, no special apparatus being required.

It has been objected that the accommodation is exercised at the same time as the convergence. I do not think that it would be advisable, in young subjects at any rate, entirely to dissociate dynamic convergence from the effort of accommodation with which it is normally always associated. But if it should seem advisable to relieve the strain on the accommodation during the exercises, this may be done in either of two ways—the patient may wear convex glasses so as to exercise less accommodation with a given amount of convergence, or, what comes to the same thing, he may wear prisms, apex in, so as to exercise more convergence with a given amount of accommodation.

These rhythmic exercises do not increase the power of the ocular muscles (any more than voice training increases the power of the laryngeal muscles), so they do not in the least diminish exophoria in distant vision. But they often much improve the power of dynamic convergence by teaching the nervous apparatus to respond more readily to the will.

In a case in which exercises have failed and the symptoms are troublesome one may, as a *pis aller,* order prisms, bases in, for near vision.

CHAPTER XII

OPERATIONS ON THE EXTERNAL OCULAR MUSCLES

THE indications for operation are fully discussed in previous chapters.

The operations commonly performed on the muscles of the eye are tenotomy, simple shortening, and advancement. Any of these measures may be employed alone, or advancement, or shortening, of one muscle may be combined with tenotomy of its opponent.

The operation of tenotomy of an ocular muscle should not be performed, either alone or in combination with advancement of the opponent muscle, save in certain quite exceptional cases. A surgeon who does not feel that he has the necessary skill and experience to perform an advancement with accuracy will best consult the interests of his patient by abstaining from operative interference in a case of squint. Many surgeons, recognising the dangers of tenotomy, but being reluctant to abandon so simple an operation, have devised modifications by means of which they hoped to minimise these dangers. One of these modifications, which appears to

have been invented by several surgeons independently at different times, consists in making a transverse cut in each edge of the muscle, the cuts overlapping but not being opposite each other; or in making transverse "button-holes" in the centre of the muscle, combined with cuts in the edges. The hope is that the cuts may gape and allow elongation of the muscle without severance of its continuity. But the muscle fasciculi and their tendinous prolongations have so little lateral cohesion one to another that if they are all divided, though at different points, they draw out from each other, and the effect is that of a complete tenotomy. If they are not all divided no effect is produced upon the position of the eye, though the mutilation may cause some weakening of the contractile force of the muscle. Twenty years ago I tried several such procedures, which were original so far as I was concerned. I have since seen equally unsatisfactory results obtained by other surgeons who have attempted tendon lengthening. Of course, all the cases have not turned out badly, any more than is the case with simple tenotomy. But the average *ultimate* results of any form of tenotomy which I have tried have been too unsatisfactory to warrant a continued employment of the operation.

Simple shortening of a muscle, by exsection of a part and reattachment to the old insertion,

is fairly satisfactory where only a very small angular effect is required, though in my hands it has not given the same uniformly good results as advancement. It is difficult to produce a large angular effect by simple shortening, and, owing to a removal of a large part of the muscle, the contractile power of the muscle and the range of rotation of the eye in the opposite direction are lessened. The same remarks apply to shortening of the muscle by folding it upon itself. There is an added disadvantage that an ugly excrescence is produced which may never quite disappear. Also, however one may scrape the surfaces of the muscle and tendon and stitch the three layers together, after a time the tuck is apt to pull out again more or less, so that partial relapses are common. I speak from experience of Todd's original muscle-tucking operation, but have not tried the modifications of it which have been from time to time described.

Advancement is the only really satisfactory and safe operation for squint.

Very many different methods of advancement of a rectus muscle have been described. I tried many of these repeatedly. None proved entirely satisfactory. Conjunctiva and episcleral tissue give no secure hold for sutures. It is not possible to produce uniformly accurate results by any of the operations in which sutures from the muscle are attached above and below the cornea, for any

difference in the tension of the sutures, whether occurring at the time of operation or during healing, will result in torsional and vertical rotation of the eye. The bulbar attachments of muscle sutures should not be spread over a greater width than the width of the muscle. In any advancement operation avoidance of unintended secondary rotation needs great care and accuracy. In advancing a muscle it is of great importance as far as possible to avoid disturbing its relations, and especially to avoid abrading its under-surface. A so-called advancement operation in which the muscle is first separated from surrounding structures at best becomes a mere shortening, for the underside of the muscle forms adhesions to its old insertion. In some cases in which this had apparently been done, without producing the desired result, I have subsequently been called upon to operate upon the same muscle. The adherent mat of muscle and scar tissue was found difficult to deal with.

The operation which I use is a true advancement; adhesions are not formed between the under-surface of the muscle and its old insertion. This I have proved when operating a second time upon the same muscle in some cases in which I had produced too large or too small a result.

Though I consider this operation to be now technically perfect, of course one does not suggest that it is not possible to produce good results by other advancement operations. A painter needs

the best pigments procurable, but can get on well with a lower quality, for these go a very small way towards the making of a picture. The skill and experience of an operator are of vastly more importance than the method of operating. In a squint operation (given adequate nursing, which one may always command in private practice) the result depends entirely upon the surgeon, who should accept responsibility for it.

The Author's Advancement Operation.

In this operation a firm unyielding hold is got for the sutures at each end, so that any desired degree of rotation of the eyeball may be produced. It admits of great delicacy of adjustment. Torsional displacement of the eye can be avoided. The anatomical relations of the advanced muscle are disturbed as little as possible. As the middle part of the muscle is not included in the sutures, its main blood-supply is not interfered with. The immediate effect produced is the final result.

Instruments.—The instruments required are: Speculum (fig. 29); straight, blunt-pointed scissors (fig. 30), a curved pair also is sometimes convenient; fixation forceps with projecting teeth which take a firm hold of the sclerotic (fig. 31); a second fixation forceps for the use of the assistant; advancement forceps (fig. 32); needle-holder (fig. 33); needles and thread. The small curved needles which I use are supplied by Messrs. Weiss

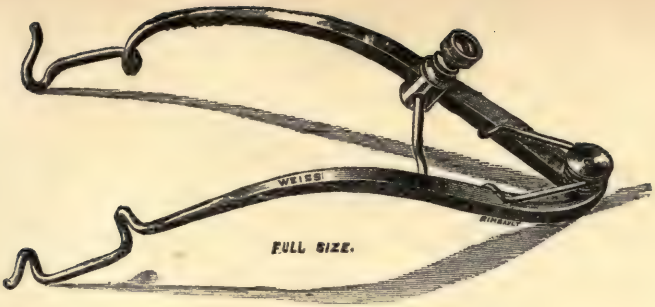

FIG. 29.

FIG. 30.

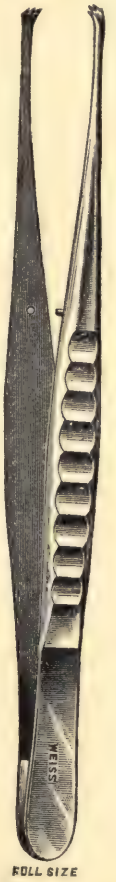

FIG. 31.

FIG. 33.

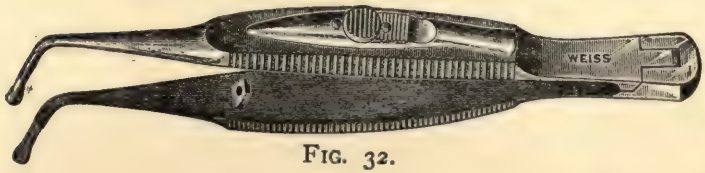

FIG. 32.

and Son. The jaws of the needle-holder are curved transversely to fit these needles. Flat jaws, however small, are apt to break a curved needle. My advancement forceps, being probe-pointed, is easy to introduce. Both blades being alike, only one forceps is required instead of two. Before use, the two blades are taken apart so that the upper blade shall not get in the way, while the lower blade is introduced as easily as a tenotomy hook. The upper blade is then attached and clamped. The short stout stem and the sliding clamp ensure a quite secure hold.

The eyes of new *needles* may require clearing with the point of a sewing needle. I formerly used only new needles, to ensure their being as sharp as possible. I find, however, that it is quite easy to sharpen them on a fine oiled stone: even new needles may sometimes be much improved. Take a needle in the holder, apply its convex side, near the point, to the stone and draw it backwards along the stone. The needles should be examined with a lens and tested on a kid drum. Any which show the smallest speck of rust or roughness on the surface should be thrown away. Needles should be kept in a book of well-washed flannel; the paper in which they are sold is apt to injure delicate points.

For the *sutures,* I formerly used black silk, which was impregnated with wax and vaseline. Even thick dyed silk, however, proved to be of

uncertain strength, and sutures prepared in bulk long beforehand were difficult to keep sterile. The object of the lubrication is to make the sutures less absorbent and to enable them to glide through the delicate structures with a minimum of damage, and of the black colour to facilitate removal afterwards.

I now use thinner undyed silk. Each needle is threaded with about 8 inches of the silk, which is then wound round a piece of lint of about the size of a postage stamp, and the point of the needle is thrust into the lint before boiling. Immediately before use, each needle and thread is anointed with sterile vaseline.

All instruments and sutures are sterilised by boiling for five minutes in distilled water which contains a little washing soda.

Dressings.—Pieces of soft linen or gauze, about 2 inches square, will be needed as swabs during the operation. A bowl of sterilised saline solution will also be required. To dress the eye on completion of the operation, I prefer circular pads about $2\frac{1}{2}$ inches diameter, consisting of many layers of plain absorbent gauze. Instead of a roller bandage, I use the Moorfields cataract sling (figs. 34 and 36), which is made of linen and tape. It holds the dressing securely, and allows it to be changed without the patient's head being lifted from the pillow. It is more comfortable and cooler than a bandage.

Preparation of the patient.—It may be advisable, for a few days before the operation, for the patient to leave off any glasses which he may have been wearing, or perhaps to wear a shade over one eye. These points are discussed in previous chapters.

The patient should be in good health. The bowels should act very freely on the day before operation. The lid margins and the conjunctival and lacrymal sacs should be clean, or the operation should be postponed until they have been success-

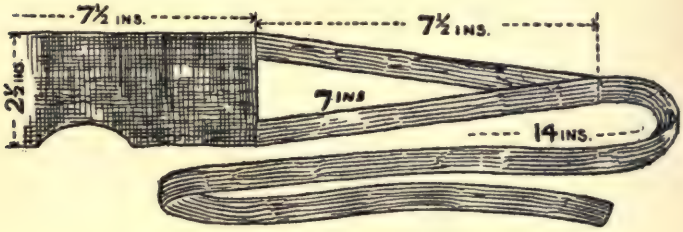

FIG. 34.

fully treated. If there be any doubt about this a culture from a swab specimen should be made and examined microscopically.

In addition to ordinary ablution, the skin of the face in the neighbourhood of the eye may be cleansed with ether soap and hot water, or swabbed with tincture of iodine. The edges of the lids are gently squeezed, and rubbed with a cotton swab. The conjunctival sac is then thoroughly flushed with warm, sterile, normal saline solution. If the operation is to be per-

formed under general anæsthesia the cleansing is better deferred until the patient has lost consciousness.

Anæsthesia.—In operating under local anæsthesia, the relative positions of the eyes can be tested while the sutures are being tightened. Under general anæsthesia this is not possible. For this reason formerly I did not care to use a general anæsthetic if it could possibly be avoided ; at present I have no such objection. The angle of deviation has, of course, been measured ; and the appearance of the eyes is studied immediately before operation. After long practice one learns to gauge the effect required by a sort of instinct, just as a violinist touches the strings at the right spots though he has no marks to guide him.

For children, general anæsthesia is required. I prefer chloroform, or a mixture of chloroform and ether : otherwise I usually leave the question of general or local anæsthesia to the choice of the patient. As a local anæsthetic, I use powdered cocaine hydrochloride applied to the conjunctiva four times at intervals of about three minutes. Before applying the first dose of cocaine, and after the last dose, and once or twice during the operation, a little adrenalin chloride solution (1 in 1,000) is dropped into the eye. If a general anæsthetic is being used, the first dose of adrenalin chloride is instilled as soon as the patient has lost consciousness.

The operation.—I prefer good daylight, but on a dull day artificial light may be used. During insertion of the sutures in the sclerotic, it is helpful to have a bright light focussed with a lens upon the site. I generally use an electric operating lamp at this stage only.

Linen mouth-masks are worn by the surgeon, his assistant, and anyone else, such as the anæsthetist, who may have occasion to speak in the immediate neighbourhood of the patient's face.

The patient lies on the table in a good light with his feet towards the window. His lids are held open by the speculum. The surgeon, standing behind the patient's head, grasps the conjunctiva with a toothed forceps, while, with the scissors, he makes a curved vertical incision through it rather more than half an inch long. The convexity of the incision is close to the corneal margin. A similar incision is made through the capsule of Tenon. The conjunctiva and capsule then retract, or, if necessary, they are pushed back so as to expose the insertion of the tendon. One blade of the advancement forceps is now entered well below the lower margin of the tendon, and passed under the tendon after the manner of a tenotomy hook. When it is accurately in place the other blade of the forceps is attached, being superficial to all the structures, so that tendon, capsule of Tenon, and

conjunctiva are all firmly held together with their relations undisturbed, except for the retraction of the membranes. The tendon, and a few little fibrous bands beneath the tendon, are now divided with the scissors at their insertion into the sclerotic. The part of the sclerotic near the cornea which is intended for the new insertion of the advanced muscle is now carefully cleared with toothed forceps of all loose tissue; for, unless the end of the muscle is held accurately in contact with bare sclerotic, there will be no firm union. The advancement forceps holding the tendon, capsule, and conjunctiva can now easily be lifted up so as to get a good view of the underside of the muscle.

It is very important to avoid any vertical or torsional displacement of the eye after the operation. In order to mark the proper alignment of the muscle during the subsequent manipulation, a suture (not shown in the diagram) is passed from the conjunctival surface through the centre of the muscle to its under-surface. It is carried under the advancement forceps to the corneal margin, where it is inserted into the sclerotic in exact alignment with the old insertion of the tendon. It is merely a marking suture, and is not intended to bear any tension, though, when tied at the end of the operation, it helps to keep the edges of the wound in accurate apposition.

Each needle and suture before being inserted is drawn through a fold of lint on which is a little sterile vaseline. One of the needles is passed inwards at A′ through conjunctiva, capsule, and muscle. It is then again passed through muscle, capsule, and conjunctiva, and brought out at B′. The bight of the thread thus encloses a width of about 2 mm. near the upper edge of the muscle, together with capsule and conjunctiva. The ends of the thread from A′ and B′ are then crossed over, making a half-hitch at C. The end bearing the needle is then entered at D and passed through conjunctiva, capsule, and muscle, and carried beneath the lower blade of the advancement forceps, out of the wound, and stuck into a piece of gauze which has been placed on the patient's forehead to receive it. The suture A, B, etc., at the lower margin of the muscle is then similarly dealt with, its needle also being brought out of the wound and stuck into the gauze.

The anterior parts of the muscle and capsule and conjunctiva are then removed by cutting them through with scissors behind where they are grasped by the advancement forceps. The longitudinal position on the muscle of the loops A, B, C, and A′, B′, C′, and the amount of tissue removed, vary according to the degree of rotation required. If only a very small effect is required, perhaps no tissue at all may be removed. The

sutures on the muscle are placed 2 or 3 mm. behind the cut end, in order that there shall be no tension on the extreme end of the muscle while union is taking place.

The next stage in the operation, the insertion of the two main sutures into the sclerotic at G

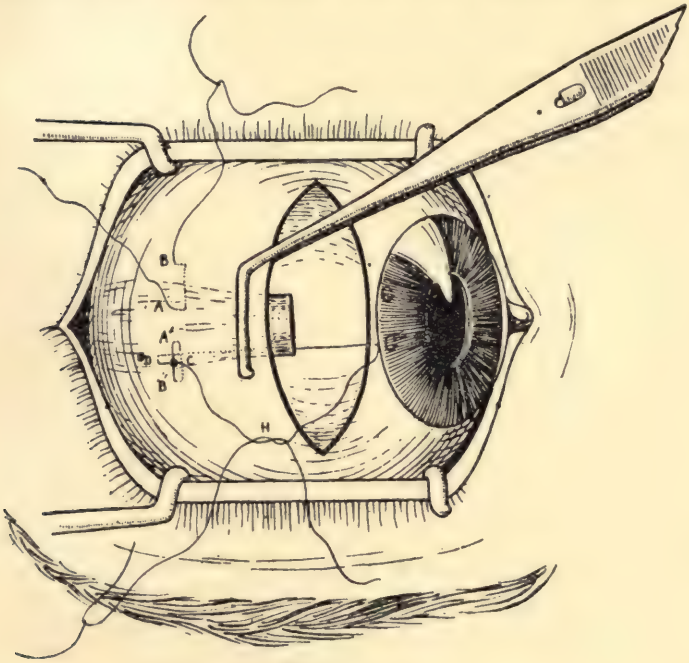

FIG. 35.

and G is one that requires great judgment and delicacy of touch. Take one of the needles in the holder, leaving about $\frac{1}{4}$ inch exposed. With the fixation forceps take a firm hold of the globe at the site of the old insertion. Lay the back of the needle on the sclerotic exactly in line

with the position of the suture in the muscle (in other words, keep the suture parallel with the "marking suture") and about $\frac{1}{8}$ inch or a little more from the corneal margin. Press the point of the needle backwards slightly, so as to dimple the sclerotic a little. Then push it onwards so that it traverses at least half the thickness of the sclerotic, but taking the greatest care not to pierce the whole thickness. The superficial extent of the insertion of the suture is about $\frac{1}{8}$ inch. A fresh hold of the needle is now taken with the holder, and the needle is pushed through a little farther, until it can be grasped again with the holder just below the point and pulled through. The other main suture is similarly dealt with. No verbal directions can be given for gauging the depth. One judges by sight and touch. One's tactile sense also warns one of an abnormal thinness of sclerotic, which is occasionally, though rarely, met with.

I now use a split-eyed needle with cutting edges for inserting the sutures into the sclerotic, but consideration of this is postponed to a later page in order to avoid complicating the description of the operation.

The assistant grasps the eyeball with fixation forceps on the opposite side, and rotates it into the primary position. The surgeon ties each suture at H and H, with the first hitch of "the surgeon's knot," gradually tightening it as he manipulates the anterior end of the muscle with

forceps into the desired position. Care must be taken that the anterior end of the muscle be neither inverted nor everted. The assistant now lets go his hold of the eye. The tension of the sutures is adjusted and each knot completed by a second hitch. These two sutures bear all the weight. The marking sutures are now tied. If there be any gaps in the membranes above or below the holding sutures, they are closed with additional fine sutures.

In operating under cocaine, before the knots are tied at H and H, the assistant holds the globe in the primary position with forceps, while the patient is told to try to look away from the operated muscle. This relaxes the muscle while it is being drawn forward by the sutures. The sutures having been temporarily secured at H and H by the first hitch of "the surgeon's knot," the assistant releases the globe. The fine adjustment is done by tightening or loosening the hitches at H and H, the result being checked by the mirror test or by the reflection of a candle flame on the corneæ. The knots at H and H are then completed.

The immediate effect is the permanent result. No over-correction, therefore, is necessary.

After the operation the eye is irrigated with sterile saline solution, and a little boric ointment is smeared on the edges of the lids. A gauze pad is applied over each eye and a strip of gauze over both pads to keep them together. The

whole is kept in position by a Moorfields sling (figs. 34 and 36).

The sutures must have a firm hold in the sclerotic. No other mode of attachment can be depended upon. In my early operations I had many failures owing to the sutures having an insufficient hold in the sclerotic. Very gradually a deeper position was

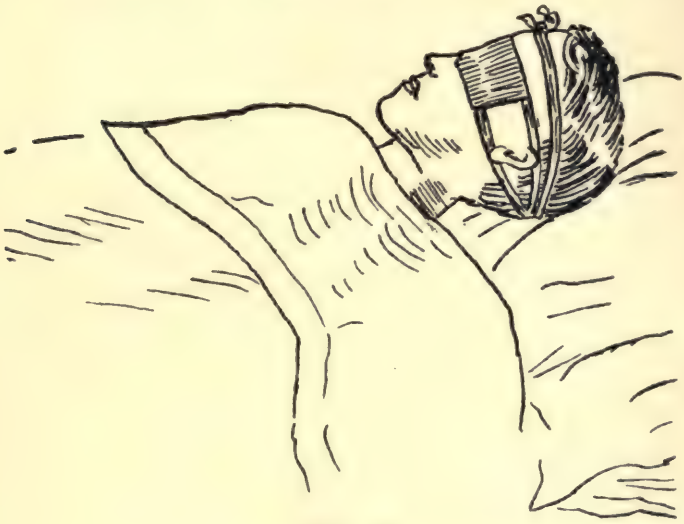

Fig. 36.

used until a certain depth was found which has proved safe and has always given a secure hold. Desiring to ascertain by inspection what proportion of the thickness of the sclerotic was traversed by the sutures, I inserted my usual advancement sutures into the sclerotic of an eye which was about to be excised for malignant disease, and Mr. Greeves, who was at that time pathologist at Moorfields,

kindly cut sections of it. My sutures traverse exactly three-fifths of the thickness of the sclerotic. This exact depth, which I have used for many years without accident, is certain and safe. But if a suture were inserted through the whole thickness of the sclerotic it would probably lead to loss of the eye ; and I have heard of such catastrophes.

Accurate insertion of these sutures is rendered unnecessarily difficult by the large expenditure of force required to make an ordinary eye-needle traverse about an eighth of an inch of scleral substance. The sclera may easily be cut with a sharp instrument ; it may be torn with great difficulty, but

FIG. 38.

it cannot be stretched to any appreciable extent. A needle, sharp at the point but not at the edges, can only pass through scleral substance by a tearing action like that of a wedge. Messrs. Weiss made me a cutting needle. The needle is straight, $\frac{3}{4}$ inch long, and has a split eye. The shaft is round. The head is trowel-shaped, each edge being as sharp and as carefully finished as the edge of a Graefe knife. The head is of such a width as to cut a slit in the sclera through which the round shaft of the needle and the suture will just pass easily. The needle is straight, because the instrument-maker has found it impossible to make perfect edges to a curved needle, owing to the difficulty in holding it. The split eye

is to enable one to use curved needles for the muscle. A straight needle cannot well be used on the lateral recti muscles of a deeply set eye nor upon the inferior or superior recti in any eye. The needles are kept in a wooden case made to hold them separately, so that the heads do not touch anything, and they are sterilised in absolute alcohol, so that no one need touch them but oneself.

For inserting the sutures in the muscle, the small curved eye needles are used. Then, as each suture is to be inserted in sclerotic, the curved needle is removed and the suture slipped into the split eye of the scleral needle. One must remember to keep the trowel-shaped head of this needle parallel with the surface of the sclerotic.

A surgeon inexperienced in advancement, before attempting the operation upon a human eye, should practise long and assiduously upon pigs' eyes until he can insert the sclerotic sutures with certainty to an exact and uniform depth. Then, in order to allow for the different thickness of the human sclerotic, he should insert the sutures in at least one human eye which is about to be excised for injury or disease, and should make sections of the eye, in order to note their position. After assisting at the operation of advancement, he will be justified in beginning to acquire experience of the operation himself.

In the foregoing description, one has had in mind advancement of one of the lateral recti. Advancement of the inferior rectus may be similarly performed, but in the case of this latter muscle perfect accuracy is even more essential

than in the case of the lateral recti. Especially is this the case if the operation is performed for hyperphoria. It is, fortunately, very rarely necessary to advance the superior rectus muscle. I have never had occasion to perform the operation except in some cases in which the superior rectus had been tenotomised for the correction of a true or apparent upward deviation. Owing to the distance of the insertion from the cornea, the prominence of the brow, and the connection between the superior rectus and the levator palpebræ superioris, the operation is difficult to perform with accuracy. In one case in which an eye turned downwards as a result of a tenotomy which a surgeon had performed some years before for the relief of an upward deviation, as the palpebral fissure was rather small, I found it necessary to divide the external canthus in order to gain room. After the operation the cut was, of course, closed with a stitch.

After-treatment.—In order to ensure an exact and permanent result, I find it necessary to keep the patient as still as possible in bed, with both eyes bandaged, for ten days,[1] at the end of which time

[1] Some surgeons have recommended a considerable over-correction at the time of operation (one writer says, "I always get as much rotation of the eye as I can") in the expectation of a partial relapse and the hope that the final result may be somewhere about what is required. If one were content with this, there would be no need to keep the patient in bed at all.

union will be quite firm. A shorter time was formerly thought sufficient, but there were some relapses which have since been avoided. In private practice a special night nurse remains in the room constantly.

The eye is dressed every day. The tapes are untied: the sling is gently lifted up from the dressing and passed upwards on to the patient's forehead. If the gauze sticks at the first dressing, it is soaked in warm boracic lotion until it drops off. A little warm lotion is allowed to run in between the lids. Vaseline is applied to the edges of the lids. Another gauze dressing is applied, and the sling is replaced without lifting the patient's head from the pillow. During the first few hours after operation, movement on the part of the patient is not likely to do harm. During the next few days, when the structures may have become slightly softened and œdematous, though there is no danger of the stitches actually giving way, any violent contraction of the ocular muscles may upset the adjustment sufficiently to prevent a perfect result.

The stitches are removed on the tenth or eleventh day. After instilling a few drops of adrenalin and 5 per cent. solution of cocaine hydrochloride, a speculum is inserted, or the lids may be held open by the fingers of an assistant. A bright light is required. One of the knots is picked up with the forceps (fig. 41) and lifted a little from the eye, and the almost invisible white suture is snipped through

beneath it. Very great care must be taken not to cut any of the muscle fibres. This is repeated for each suture. If only one eye is to be operated upon no further dressings are required, and the patient may get up and go out. If it has been decided to operate upon both eyes, the second eye is operated upon ten days after the first, and the sutures are removed from the first eye at the same time.

Confinement to bed with both eyes bandaged for ten or twenty days is not found nearly so tedious as one might think, either by children or by adults, and the results amply repay the surgeon for the extra trouble involved.

Secondary advancement.—Occasionally a patient presents himself with one eye widely divergent as a result of tenotomy of an internal rectus muscle. Usually, an excellent cosmetic result may be obtained by advancing the retracted muscle, even after many years. One often finds the muscle in surprisingly good condition, even though it may have little or no attachment to the globe. Sometimes, however, it may be more or less atrophied from disuse.

The conjunctiva and capsule of Tenon may be matted to the globe in the neighbourhood of the former insertion of the muscle, or the eyeball may be quite destitute of covering in this region. Grasp the membranes with toothed forceps well above or below the scarred area, and, with the

scissors, separate them from the nasal side of the eye. A squint hook may be of assistance in this. If the muscle is not found attached to some portion of the anterior segment of the globe it is of no use searching for it with a squint hook far back. It is never found attached to the posterior hemisphere. Seize the coverings of the eye in the region of the sunken caruncle, pull them forward, lift them up. The muscle, or what remains of it, will be seen on their under surface. I think that, after tenotomy, the tendon fails to become reattached directly to the globe far more often than is generally supposed. The muscle and membranes are seized with toothed forceps and drawn between the jaws of an advancement forceps. The surface to which they are to be attached must be well refreshed.

Tenotomy.

The instruments required are speculum (fig. 29), straight blunt-pointed scissors (fig. 30), tenotomy hook (fig. 40), and fixation forceps (fig. 31). The patient lies on a table. The eye to be operated upon is cocainised and is irrigated with sterile saline solution, and supra-renal extract is instilled. In operating on the left internal rectus it is more convenient to stand in front and on the left of the patient, and in tenotomising any of the other recti to stand behind the patient's head. The speculum is inserted. Tell the patient to look

in a direction opposite to that of the tendon to be divided, so as to bring its insertion well forward. With the forceps, pick up the conjunctiva over the

FIG. 39.

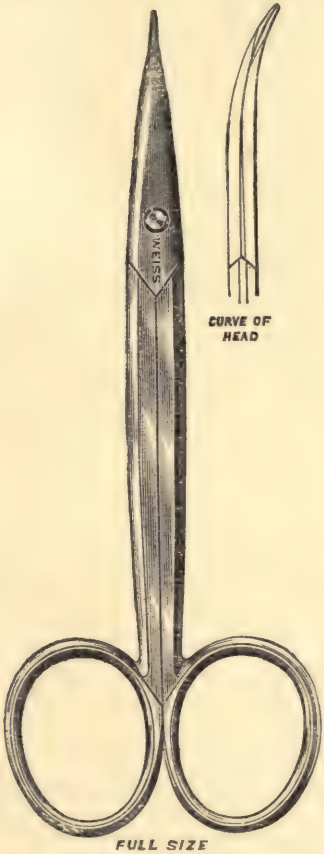

FIG. 40.

FIG. 41.

FIG. 42.

insertion of the tendon, and, with the scissors, make an incision, about a quarter of an inch long, in a direction at right angles to that of the tendon.

Now divide the capsule of Tenon in the same way. This brings the insertion of the tendon into view. While the forceps still holds up the cut edge of the capsule, make a few short snips with the scissors near one border of the tendon, until the point of the scissors is felt to slip freely back without encountering any resistance. Now lay down the scissors, and take up the hook in the right hand. Pass the point of the hook into this incision, and hook it round the insertion of the tendon, until it appears at the other border. During this manœuvre, the point of the hook should be kept in contact with the sclerotic. Now lay down the forceps and transfer the hook to the left hand. Take care to avoid any dragging with the hook, as this causes pain. With the scissors, cut between the point of the hook and the globe, until the tendon is divided at its insertion and the hook comes away. It is usual to reintroduce the hook, to seek for any fibres of insertion which may have escaped division. Spare the lateral expansions even though the result of the tenotomy appear insufficient. It is not necessary to suture the conjunctiva, unless the conjunctival incision is unusually large.

After the operation, there is a very considerable defect of movement in the direction of action of the tenotomised muscle. In many cases this, to some extent, subsequently disappears.

A pad and bandage should be worn for the first

twenty-four hours, after which it may be discarded. The eye should be bathed with boric lotion, three or four times a day, until the wound is healed.

Complete central tenotomy.—In America, partial tenotomies are very commonly performed. I believe these to be useless, as, until the whole of the tendon proper has been divided, no appreciable effect is produced. But by the neat method by which these partial tenotomies are performed, the tendon itself may be completely divided, leaving its lateral expansions absolutely intact.

The patient is prepared as for an ordinary tenotomy. The forceps, scissors, and tenotomy hook required are those of Stevens (figs. 42, 40, and 41). The surgeon, standing behind the patient's head, seizes, with the forceps, the conjunctiva over the tendon near its insertion. A transverse incision of sufficient length is made through the conjunctiva. The capsule of Tenon is now similarly incised to an extent just sufficient to expose the tendon. The central fibres of the tendon are next grasped with the forceps, and a button-hole is snipped through the tendon with the scissors. The forceps being laid aside, the small tenotomy hook is introduced through this button-hole, with its point turned towards one border of the tendon. Half the tendon is cautiously snipped through on the hook, from the centre towards the edge. The point of the

hook is now turned round, and the other half of the tendon similarly divided.

When the hook is first introduced through the button-hole, on lifting it up the tension of the tendon is felt. When the whole of the tendon proper has been divided, considerable tension is still felt, by means of the hook, at the edges of the wound. This resistance is due to the lateral expansions of the tendon. These should on no account be divided, as it is upon the preservation of these lateral expansions that the comparative safety of this operation depends. Sometimes it is impossible to define exactly the edge of the tendon, owing to its gradually merging into its lateral expansions. In such a case one has to be guided by the degree of tension felt by the hook.

No after-treatment is required beyond frequent bathing with boric lotion or sterile saline solution.

As a rule the effect produced is small—about 6° or 7° in the case of the internal rectus. What has been said about the dangers of ordinary tenotomy applies to this method also, though in rather less degree (*e.g.*, see Case A, 541, p. 160).

APPENDIX

CONGENITAL AMBLYOPIA

Table vi. shows the degrees of refractive error and the visual acuity in the twenty-three cases of congenital amblyopia described in chapter v.

TABLE VI.

BETTER EYE.			WORSE EYE.		
Refractive Error.		Vision.	Refractive Error.		Vision.
Lower Meridian.	Higher Meridian.		Lower Meridian.	Higher Meridian.	
+1	+1	6/6	+2·5	+4	6/60
−0·5	0	6/6	−0·5	+4·5	6/36
+1·5	+1·5	6/6	+1	+3·5	6/24
−1·5	+2	6/9	−2	+2·5	6/18
+1	+2	6/6	+2	+5	6/18
+1·25	+2	6/6	0	+5·25	6/36
+1·25	+1·25	6/6	+5	+7	6/36
+1·5	+1·5	6/6	+5	+6·5	6/24
+4	+4	6/6	+5·5	+8·5	6/24
+0·5	+0·5	6/6	+1	+1	6/36
+0·5	+0·5	6/6	+4	+6	6/60
+1	+1·25	6/6	+1	+4·5	6/24
+3·5	+3·5	6/6	+2	+6·5	6/36
+1·5	+1·75	6/6	+1·5	+4·75	6/18
+0·5	+0·5	6/6	−1	+3	6/24
+1	+1	6/6	+1	+4	6/24
0	0	6/6	+4	+6·5	6/24
+3	+3·5	6/9	+3	+7	6/36
+1	+1	6/6	−1·5	+2·5	6/24
+0·75	+1	6/6	+3	+5·5	6/18
+2	+2	6/6	+2	+6·5	6/36
+0·5	+0·5	6/6	+1	+4·5	6/18
+0·25	+0·75	6/6	+1·5	+5·5	6/24

Table vii. shows the refractive error and the visual acuity of the eleven cases in table iii., chapter v., which had an amblyopia of $\frac{6}{18}$ or higher. The visual defect in these cases also is probably congenital.

TABLE VII.

Fixing Eye.			Deviating Eye.		
Refractive Error.		Vision.	Refractive Error.		Vision.
Lower Meridian.	Higher Meridian.		Lower Meridian.	Higher Meridian.	
+2	+2	$\frac{6}{6}$	+1·5	+5·5	$\frac{6}{24}$
+3·5	+4	$\frac{6}{6}$	+3·5	+6	$\frac{6}{18}$
+1·5	+1·5	$\frac{6}{6}$	+1·5	+5	$\frac{6}{24}$
+4	+4·5	$\frac{6}{6}$	+2	+5·25	$\frac{6}{18}$
+2	+3	$\frac{6}{6}$	+2	+7	$\frac{6}{36}$
+1	+1	$\frac{6}{6}$	+1	+3·5	$\frac{6}{18}$
+2·5	+3	?	+0·5	+3·5	$\frac{6}{18}$
+3	+3	$\frac{6}{6}$	+3·5	+7·5	$\frac{6}{60}$
+0·5	+0·5	$\frac{6}{6}$	−1	+2·75	$\frac{6}{18}$
+2·75	+3·5	$\frac{6}{6}$	+1	+5·5	$\frac{6}{24}$
+1·25	+1·25	$\frac{6}{6}$	+4·5	+8·5	$\frac{6}{24}$

PRISMS AND DECENTRED LENSES.

There are several systems of numbering prisms. In ordering a prism it is necessary therefore to specify which system one uses. In this book the strength of a prism is expressed by the number of degrees which it deflects a ray of light. This "deviating power" is about half the geometrical angle (the angle between the two plane surfaces).

A prism causes mal-projection, and chromatic dispersion of white light. Clinically, mal-projection is to a great extent avoided by dividing the prism between the two eyes. For instance, in a case of right hyperphoria 2°, if one wished to correct $1\frac{1}{2}°$ of the error, one would order a prism $\frac{3}{4}°$ base down, before the right eye, and a prism of the same strength,

base up, before the left eye. Chromatic dispersion is not noticeable in a prism which does not exceed 2° or 3° deviating power. Prisms with bases towards the temples often give rise to annoying multiple reflections.

A pencil of parallel rays which traverses a spherical lens at its optical centre is made convergent or divergent. A pencil of rays, traversing a lens towards its periphery, is deflected in addition (as by a prism) towards the axis of a convex lens or away from the axis of a concave lens.

Glasses containing prisms in combination with spherical and cylindrical lenses are expensive, because they must be specially ground—the optician cannot prepare them from his stock. But if the patient has to wear fairly strong lenses, and if the required prismatic effect is small, this may be secured by decentring the spectacle glass. That is, the optician, instead of cutting the spectacle glass from the middle of one of his ready-ground lenses, cuts it from one side. This is much cheaper. The effect of decentring a cylindrical lens, in a direction at right angles to its axis, is the same as that of decentring a spherical lens. In a lens of the ordinary stock size there is room to decentre a medium-sized spectacle glass about 4 or 5 mm., *i.e.*, a total of 8 or 10 mm. in the two eyes.

The following table, prepared from Dr. Maddox's formula, shows the prismatic effect of decentring lenses.

	2 mm.	3 mm.	4 mm.	5 mm.	6 mm.
2 D	14′	21′	27′	35′	41′
3 D	21′	31′	41′	52′	1° 2′
4 D	27′	41′	55′	1° 10′	1° 22′
5 D	35′	52′	1° 10′	1° 26′	1° 43′
6 D	41′	1° 2′	1° 22′	1° 43′	2° 4′
7 D	48′	1° 12′	1° 36′	2°	2° 24′
8 D	55′	1° 22′	1° 50′	2° 19′	2° 45′

Results of Advancement Operations.

The following statistics of 100 *consecutive cases of advancement, beginning January,* 1899, *are now of course much out of date. On the whole, the average results were good. I leave them because they are instructive. Twenty years' further experience and practice have enabled me to operate with far greater certainty and accuracy, but, however gratifying to oneself it might be to record greatly improved results, they might be misleading as a guide.*

In performing advancement I have, since 1898, relied *exclusively* upon the method described in chapter xii. I therefore give results of 100 consecutive advancement operations, beginning January, 1899. I have re-examined most of these cases within the present year (1906).[1]

By *primary advancement* I mean advancement of a muscle which had not previously been operated upon. In some of these cases the opposing muscle had previously been tenotomised by some other surgeon. By *secondary advancement* I mean operation upon a muscle which had previously been tenotomised or unsuccessfully advanced.

In cases in which the fusion sense has been fairly well developed, but in which the deviation persists, the object of operation is to put the eyes into such a position that the patient will have binocular single vision. If the fusion sense cannot be developed, one aims at removing the obvious deformity.[2] Cases which come up to this standard I have classified as

[1] And on many subsequent occasions also.

[2] I am now more exacting in this respect and do not consider a case successful unless movements are full in all directions and the mirror test (p. 80) shows that the eyes really are straight. On going through the notes of the 66 cases which were considered successful, I find that 13 fall short of perfection as judged by my present standard.

successful. Those which do not I have called unsuccessful, although in all these cases there was considerable improvement.

Eighty-six operations were performed under cocaine and 14 under chloroform.[1]

The 100 cases consisted of :—

Primary	advancement of	external	rectus ...	77
,,	,,	internal	,, ...	8
,,	,,	inferior	,, ...	1
Secondary	,,	external	,, ...	4
,,	,,	internal	,, ...	9
,,	,,	superior	,, ...	1

The 77 cases of primary advancement of external rectus included one case of congenital paralysis of external rectus and two cases in which the muscle was atrophied. In these three cases, though a perfect result was not to be expected, musculo-capsular advancement, combined with tenotomy of the opposing muscle, produced considerable improvement in the appearance.

Of the 74 remaining primary advancements of external rectus, 66 were successful; in three other cases a second operation[2] proved successful; in one a second operation was recommended but was refused; in four cases, though the results did not satisfy me, the patients were quite satisfied, so, as there was no

[1] Formerly I seldom ventured to operate unless the operation could be done under local anæsthesia (see p. 213). I have now no objection to the use of a general anæsthetic.

[2] I have not included these three cases in the list of secondary advancements recorded below.

In two of these the result of the first operation had been insufficient, and in the other case it had been excessive. In each case I had the opportunity, during the second operation, of proving by dissection that my advancement operation is a true advancement of the insertion of the muscle. A tenotomy hook passed under the muscle could be brought freely forward to the new insertion, showing that no adhesions had formed to the original insertion. This is a very important point.

fusion sense in any of these four cases, I did not urge further operation. In 17 of these 74 cases the internal rectus was tenotomised at the same time.[1]

Of the eight primary advancements of internal rectus, two were old myopic divergent squints, and two were neuropathic divergent squints with some fusion sense. These were successful. The remaining four were neuropathic divergent squints with no fusion sense. Two of these were successfully operated upon. Considerable improvement in appearance was produced in the remaining two cases, but the results were far from perfect. In four out of the eight cases both interni were advanced. The external rectus was not tenotomised in any case.

The primary advancement of inferior rectus was in a case of vertical deviation of high degree with absence of fusion sense. I succeeded in removing the deformity.

The four secondary advancements (readvancements) of external rectus were in cases in which someone had unsuccessfully attempted advancement. In one case I succeeded. In the other three the previous mutilation[2] of the parts rendered a perfect result impossible.

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[1] Of the 17 cases of combined tenotomy, four were followed by divergence of the eye beginning from five to twelve years after the operation. In each case, secondary advancement of the tenotomised muscle proved successful. I do not now perform tenotomy even in combination with advancement (see page 116).

[2] In a case in which a suture has been tied on the muscle, *including its whole width,* the muscle in front of the ligature atrophies just as surely as if it had been cut off with scissors.

If a surgeon in advancing a muscle has isolated it from its overlying membranes and its lateral expansions, if one has to operate again upon this muscle one finds a shapeless mat of muscle and scar tissue.

The unsuccessful previous attempts at advancement in these three cases had all been made by the same surgeon, who committed both these mistakes.

The nine secondary advancements of the internal rectus were all cases in which tenotomy of this muscle had been followed by divergence of the eye. I was responsible for two of these tenotomies. Five of these secondary advancements were successful; in three the condition was improved; in one there was no improvement.

The secondary advancement of the superior rectus has already been described (Case B, 227, chapter x.).

The Deviometer (p. 89).

The patient's eye is only two feet from the instrument. In looking at the button above the light he will, therefore, exercise a dynamic convergence proportionate to that distance. It has often been suggested to me that this would cause the degree of a convergent squint to appear greater than it really is. This is not the case, because the surgeon's eye is at the same distance, just above the zero of the scale. This is easily demonstrated by experiment with a normal-sighted person. When he looks at the button both visual axes converge to this point. The surgeon places his eye at the same distance—just above the button—so that he sees the vertical lines of light occupying symmetrical positions on the corneæ of the observed person, showing that the latter has no squint. If the observed person could look at the button with one eye, without exercising any dynamic convergence (keeping his visual axes parallel), he would appear to the surgeon to have a divergent squint—in fact, he *would* have a divergent squint for that distance.

No adjustments are required except that the 60 cm. string must be kept taut. As the scale is flat, instead of a curved arc, a slight lateral movement on the part of the patient introduces no appreciable error. The height of the patient's eyes above the tables makes no difference.

Details of construction.—The woodwork can be made by any carpenter for a few shillings. The following measurements have been found satisfactory :—

The pedestal, 10 inches wide, 5 inches deep, from before backwards, $2\frac{1}{2}$ inches high.

The upright board, height $13\frac{1}{2}$ inches (11 inches above pedestal), width 5 inches, thickness $\frac{3}{4}$ inch.

The arm is pivoted at one end by a bolt which passes through the upright board. There is a chock on each side, on one of which it rests. It is swung over to either side as required. This arm is of hard wood, 27 inches long, 2 inches wide, $\frac{3}{16}$ inch thick. It is painted black in front. A long strip of white paper or celluloid, about half an inch wide, has marked on it the tangents to degrees at the distance of 60 cm. This strip is pasted on the back of the arm, with the zero of the scale at the pivot hole.

The tangents to degrees at 60 cm. are—

2° – 2·1 cm.	18° – 19·5 cm.	34° – 40·5 cm.
4° – 4·2 ,,	20° – 21·8 ,,	36° – 43·6 ,,
6° – 6·3 ,,	22° – 24·2 ,,	38° – 46·9 ,,
8° – 8·4 ,,	24° – 26·7 ,,	40° – 50·3 ,,
10° – 10·6 ,,	26° – 29·3 ,,	42° – 54 ,,
12° – 12·8 ,,	28° – 31·9 ,,	44° – 57·9 ,,
14° – 15 ,,	30° – 34·7 ,,	46° – 62·6 ,,
16° – 17·2 ,,	32° – 37·5 ,,	48° – 66·7 ,,

In inserting the bell push only one wire is cut, the other being left intact.

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